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HUMAN BIOLOGY
a record of research

FEBRUARY, 1955

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VARIATIONS IN THE CARIES SUSCEPTIBILITY OF CHILDREN'S TEETH

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INTRODUCTION

CONSIDERABLE variation in susceptibility to dental decay occurs between teeth in different regions of the mouth and between various surfaces of the same tooth. The first type of variation has been studied by Knutson, Klein and Palmer ('38), and the second by Hyatt ('27, '30), Leigh ('24) and Walsh and Smart ('48). This paper presents the results of a further investigation of such differences in susceptibility, in which smooth and pitted surfaces have been examined separately since a previous study had suggested that these may differ in etiology (Grainger and Reid, '54).

The data were obtained by examining the permanent teeth of 1144 school children, ages 6 to 17 years inclusive, in the town of Burlington, Ontario. Examinations were done by the district dental health officer with mouth mirror and explorer according to a recording system already reported (Grainger and Reid, '54). The surfaces of the teeth were divided into areas which appeared to act as discrete and independent origins of dental caries. These were basically the mesial, distal, lingual, labial or buccal, and occlusal surfaces; the incisal edges were ignored. The buccal surfaces of the lower molars, the lingual surfaces of the upper molars, and the lingual surfaces of the upper incisors were further sub-divided into the smooth area near the gingiva, and the pitted or fissured region farther from the gum. The presence or absence of each permanent tooth, and the caries-affected surfaces were recorded for each child. The proportions of the specific tooth surfaces decayed of those examined were then calculated for each age, combining the two sides of the mouth. The number of missing teeth was not great enough to influence perceptibly the results obtained.

The following section outlines the statistical analysis used in the study.

STATISTICAL ANALYSIS

It seems likely that caries-producing attacks tend to occur randomly in time. If, then, the attack rate remains constant, while differences between individuals are small relative to chance variation, the proportion of surfaces free from decay, at age t, may be approximately written as

$$P_t = e^{-b(t-a)},$$

where b is a measure of the attack rate and a is the age at which exposure begins. b, in fact, could be interpreted as the number of caries-producing attacks per year. This expression implies that

$$-\log_e P_t = k + bt;$$

that is, the natural logarithm of the proportion free of decay is linearly related to age, the slope of the straight line being the caries attack rate.

The requirements for this simple relationship may be met by individual tooth surfaces for children of the ages studied. Conditions predisposing to cavitation probably do not change greatly in the mouth, nor in the local environment of the surface, over this period. While the total number of caries lesions accumulated will reflect individual variation in susceptibility, this certainly would be of much less importance for the single surface. Variation in the age of eruption of teeth would tend, naturally, to cause deviation from the above pattern. However, assuming that the distribution of eruption times is a normal distribution and the value of b is small, it may be shown that the simple relationship should hold from about two to three years after the average age of eruption, most individuals having the particular tooth involved by this time.

The application of this formula to the data for the distal surface of maxillary first premolar is illustrated in figure 1. In the upper part of the figure the proportions of surfaces free of decay are plotted from age 10 to age 17, the average age of eruption being 10.4 years. The proportion free of decay declines steadily from 12 years. In the lower part of the figure the negative logarithms of the proportions are plotted against time, giving a reasonably straight line with slope of about 0.07.

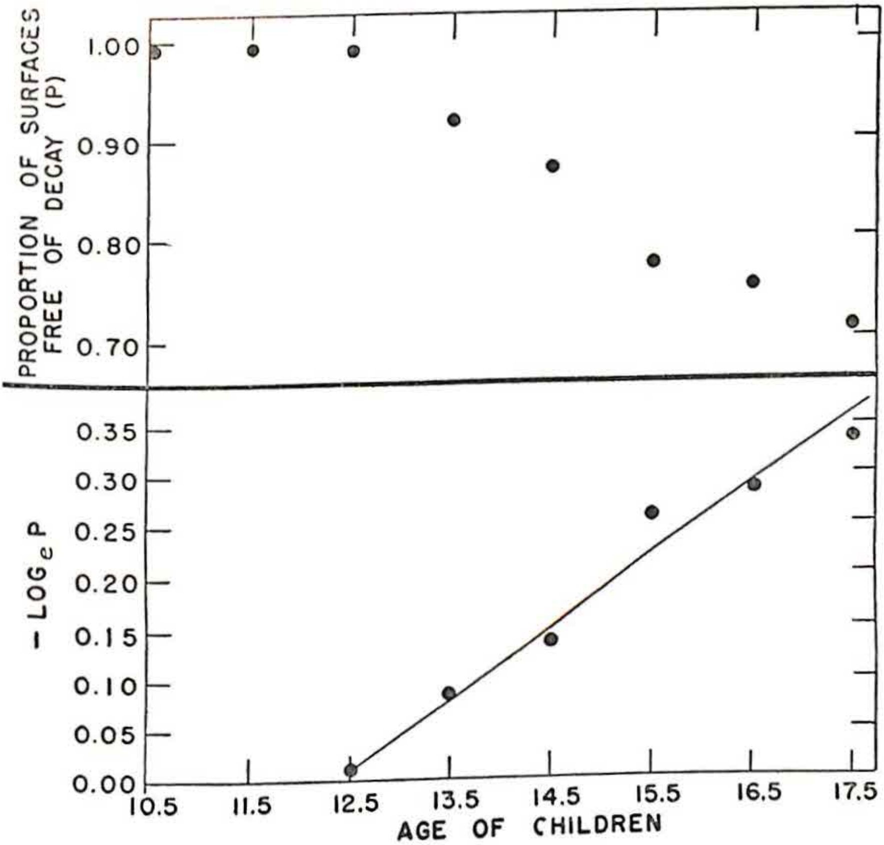

FIG. 1. THE ACCUMULATION OF DENTAL CARIES ON THE DISTAL SURFACE OF THE MAXILLARY FIRST PREMOLAR.

The surface is accumulating cavities at an average rate of 0.07 cavities per year.

This constant may be estimated arithmetically in a number of ways. Since the point of origin cannot be readily specified, it is probably best to estimate both a and b. By the method of least squares,

$$b = \frac{\Sigma W(y - \bar{y})(t - \bar{t})}{\Sigma W(t - \bar{t})^2},$$

where $y = -\log_e P$, and $W = \frac{N\hat{P}}{(1 - \hat{P})}$. $\hat{P}$ is the "true" proportion of decayed surfaces and N the number of surfaces examined at any age. Since $\hat{P}$ is not known these calculations must use weights estimated from a graphical fit of the data or a previous cycle of iteration. In addition, it is possible to evaluate

$$\chi^2 = \Sigma W(y - \bar{y})^2,$$

to serve as a measure of goodness of fit of the relationship. If the fit is satisfactory, the standard error of b is given by

$$s_b = \frac{1}{\sqrt{\Sigma W(t - \bar{t})^2}}$$

For distal surface of maxillary first premolar the method yields the estimates: $b = 0.071 \pm 0.006$; $\chi^2 = 1.52$. The value of χ^2 is based upon 4 degrees of freedom, and is clearly not significant.

It is assumed in these calculations that the individual observations are independent of one another. Since two surfaces from each individual are included in each proportion this is not strictly correct. However, on smooth surfaces at least, the correlation between the two sides of the mouth is small and would not greatly affect the validity of the procedure.

RESULTS

In most cases it was found possible to estimate satisfactorily the caries attack rates for smooth surfaces by the method outlined, though on some of the surfaces there was so little decay that no attempt was made to do so. These rates are given in table 1. In general, the susceptibility of the teeth increases posteriorly in the mouth. An additional slight increase was observed in three mesial surfaces of maxillary teeth, extending from the canine to the first incisor. Adjacent pairs

TABLE 1

Caries attack rates of specific smooth tooth surfaces

Tooth	Maxilla			Mandible			
	Labial or Buccal	Mesial	Distal	Labial or Buccal	Mesial	Distal	Lingual
1st Incisor	—*	.032 ± .003	.026 ± .004	—	.002 ± .001	.003 ± .001	—
2nd Incisor	—	.023 ± .003	.007 ± .002	—	.003 ± .001	.001 ± .001	—
Canine	—	.005 ± .003	.011 ± .003	—	—	—	—
1st Premolar	—	.020 ± .003	.071 ± .006	—	.004 ± .002	.030 ± .004	—
2nd Premolar	—	.056 ± .006	.068 ± .007	—	.027 ± .005	.037 ± .006	—
1st Molar**	.008 ± .002	.052 ± .003	.005 ± .002 .062 ± .012	.007 ± .001	.054 ± .004	.013 ± .003 .081 ± .010	.007 ± .002
2nd Molar	.026 ± .006	.017 ± .006	.010 ± .004	.017 ± .003	.035 ± .011	.011 ± .005	—

* A dash indicates that either no caries occured, or so small a number that the caries attack rate could not be determined. This was the case for all upper lingual smooth surfaces.

** The second rate for distal surfaces of the first molar refers to the period following the eruption of the second molar.

of interproximal tooth surfaces are, in many instances, quite similar in susceptibility. The buccal and labial surfaces on the whole have higher rates than the lingual, while the interproximal have much higher rates than any of these.

The distal surfaces of the first molar present an interesting variation on the general pattern. The negative logarithms of the proportions free of decay for this surface on the mandibular first molar are plotted against age in figure 2. The trend of these figures suggests that a change occurred in the average rate of accumulation of cavities about age 13, a year or so after the average age of eruption of the second molar. For this reason two straight lines were fitted, the one to age 12 inclusive and the other from age 12 to age 17. The slopes of these are 0.013 cavities per year and 0.081 cavities per year, respectively. The same procedure was needed for the upper first molar.

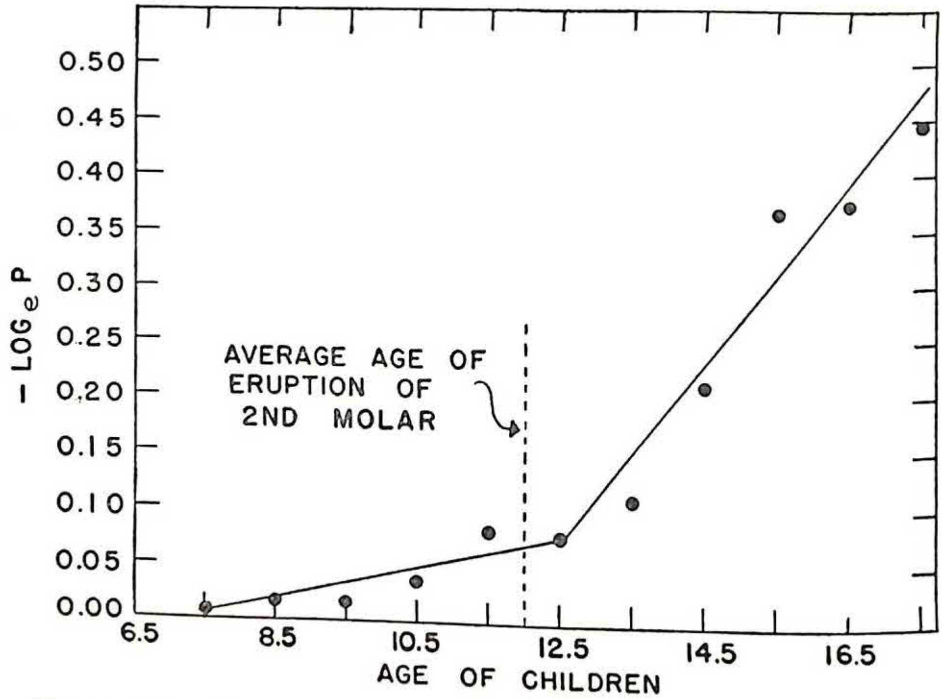

FIG. 2. THE ACCUMULATION OF DENTAL CARIES ON THE DISTAL SURFACE OF THE MANDIBULAR FIRST MOLAR.

The determination of the values of b was difficult for some of the pitted surfaces. The reason for this is evident from the decay pattern shown in figure 3 for the occlusal surface of lower first molar, which is fairly typical. Decay on this surface seems to have taken place from the first appearance of the teeth in the mouth so that the proportion free of decay declines very rapidly. However, after this initial fall the proportion remains relatively constant, as if a certain proportion of individuals are resistant to decay on this surface. There is a subsequent slow increase in the proportions, but this may be due to drilling down through the intact occlusal surface in order to gain access to a carious interproximal surface. On this assumption the proportion free of decay can be corrected so that it is based only on the group which shows decay over the initial period. In this case about 24% might be assumed caries resistant. A straight line could then be fitted to the points available, those for the years 6, 7 and 8. The value of b is 0.58. This procedure is not satisfactory since only a few points are available for the estimation of b, including one in the neighborhood of the average age of eruption. In addition considerable correlation probably exists between the occur-

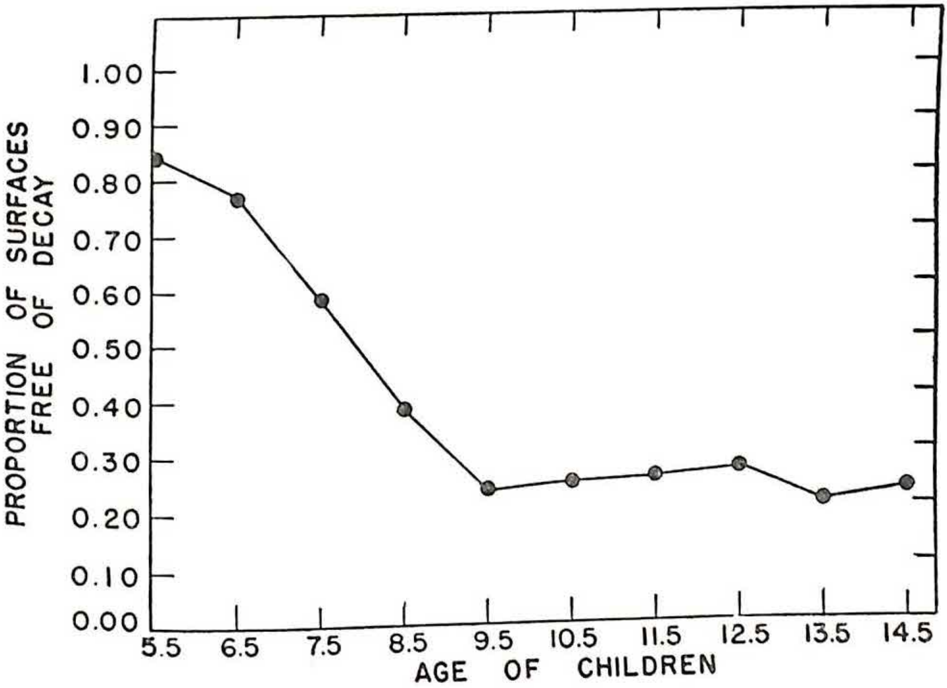

FIG. 3. REDUCTION IN THE PROPORTION OF SURFACES FREE OF DECAY ON THE OCCLUSAL SURFACE OF THE MANDIBULAR FIRST MOLAR.

rence of cavities on this surface in opposite sides of the mouth. However, the procedure does provide an estimate comparable to those for the smooth surfaces.

All of the pitted surfaces appeared to reach a maximum proportion decayed except the occlusals of the premolars, which may indicate that these should be considered as smooth surfaces. Where necessary the pitted surfaces were then adjusted on the basis of the stable period, and decay rates estimated. These are given in table 2. Standard errors are also tabulated, though these may considerably underestimate the correct values. As a group, the pitted surfaces are much more susceptible than the smooth surfaces; this is notable for the buccal or lingual pitted surfaces as compared to the smooth surfaces of the same tooth. They are somewhat more susceptible in the back of the mouth, though the difference is not nearly as marked as for the smooth surfaces.

TABLE 2

Caries Attack Rates of Specific Pitted or Fissured Tooth Surfaces

Tooth	Maxilla		Mandible	
	Occlusal	Lingual	Occlusal	Buccal
1st Incisor		—		
2nd Incisor		.29 ± .08 (9%)*		
1st Premolar	.040 ± .006		.008 ± .003	
2nd Premolar	.080 ± .008		.047 ± .008	
1st Molar	.55 ± .04 (73%)	.16 ± .02 (24%)	.58 ± .08 (76%)	.30 ± .04 (40%)
2nd Molar	.56 ± .07 (76%)	.44 ± .21 (9%)	.94 ± .21 (87%)	.71 ± .24 (20%)

* The value in parentheses is the estimated maximum percentage of surfaces susceptible, used in the adjustment of the proportion of surfaces decayed.

DISCUSSION

The greatest contrast in the attack rates shown is between those for smooth surfaces and those for pitted surfaces. The presence of a deep pit or fissure on the surface of a tooth seems to result in a cavity developing soon after tooth eruption. This occurs even though the pits are located in regions where the caries producing influences are weak. In a certain percentage of individuals decay does not occur either because the pits are not formed in the development of the tooth, or because the mouth is inherently immune. This percentage is small for the occlusal surfaces, but relatively large for some of the other pitted surfaces.

The pattern of decay on the smooth tooth surfaces seems, on the other hand, to reflect environmental variations in the mouth. The rates for interproximal surfaces are high and tend to be similar for adjacent surfaces, reflecting the local conditions of the space between the teeth. The relatively low rate of decay on the mesial surface of the maxillary second molar is an exception to the general pattern. The increased rate of decay of the distal surfaces of both maxillary and mandibular first molars upon the eruption of the second molar further suggests that the higher rates for interproximal surfaces are associated with confinement of the tooth surface. The lowest attack rates are found where the saliva is abundant near the openings of the salivary ducts, and where the action of the lips and tongue, and the masticating action of the teeth, prevent stagnation.

It is of interest to compare these data with two sets from other localities. The first were obtained by an expedition to the mountains of Greece, where villagers were living on a rather restricted diet thought to be low in minerals (Moyers and Johns, manuscript). The second set were obtained from Aylmer, Ontario, an area where the natural water supply has contained 1.2 p.p.m. of fluoride for at least 18 years (Coburn and Knipe, '53). The attack rates for the surfaces of the anterior maxillary teeth, and the mandibular first molars in these areas, together with estimates obtained in the present study, are given in table 3. These teeth were selected since they are representative of pitted and smooth surface caries on both the anterior and posterior teeth. Neither the Burlington nor the Greek water supplies contained significant amounts of fluorides.

The caries attack rates for the Greek area are quite similar to those of Burlington, Ontario. Moreover, the Greek data confirm the observation that the proportion of pitted surfaces decayed reaches a maximum very shortly after the teeth erupt. There was, however, no subsequent increase on the occlusal surfaces of the molars, probably because none of these children had dental treatment. The natural fluoride area shows an interesting departure from the pattern of decay observed in the other two areas. While the pitted surface attack rates appeared to be about the same as elsewhere, the smooth surface rates were very much lower in both the anterior and the posterior teeth. Reports on fluoride studies have frequently emphasized the protection given to the anterior teeth.

and have suggested that these teeth received additional benefit because they were bathed in water in the act of drinking. However, it seems that the smooth surfaces of the posterior region are just as well protected as those of the anterior region; the relatively lower protection afforded to the posterior teeth is due to the presence of a greater number of pits and fissures on these teeth.

TABLE 3

Comparison of Caries Attack Rates for Certain Tooth Surfaces, in three Geographical Areas

Tooth Surface	Source of Data Burlington, Ontario	 Greece	 Aylmer, Ontario
Smooth:			
Maxillary Anterior			
Mesial, 1st incisor	.032 ± .003	.020 ± .004	.008 ± .003
Distal, 1st incisor	.026 ± .004	.020 ± .003	.007 ± .002
Mesial, 2nd incisor	.023 ± .003	.020 ± .004	.012 ± .005
Mandibular Posterior			
Mesial, 1st molar	.054 ± .004	.053 ± .006	.017 ± .003
Distal, 1st molar **	.013 ± .003 .081 ± .010	.014 ± .004 .030 ± .020	— .006 ± .009
Pitted:			
Maxillary Anterior			
Lingual pit, 2nd incisor	.29 ± .08 (9%)	(4%)*	.21 ± .03 (3%)
Mandibular Posterior			
Occlusal, 1st molar	.58 ± .08 (76%)	(52%)*	.75 ± .06 (54%)
Buccal pit, 1st molar	.30 ± .04 (40%)	.22 ± .09 (27%)	.48 ± .07 (21%)

* The carries attack rates could not be estimated for these surfaces.

** The second rate refers to the period following the eruption of the second molar.

The proportions of susceptible pitted surfaces are similar in the Greek and fluoride areas, being less than the corresponding values found in Burlington. The differences may represent real differences in susceptibility in these areas, though they may be due in part to differences among examiners or in the level of treatment. In the latter case it would imply that a proportion of the "carious" pits which occur on these surfaces are not progressive in nature.

SUMMARY

The rate of caries accumulation on specific permanent tooth surfaces of school children in Ontario was estimated from the slope of the line relating the negative logarithm of the proportion of surfaces free of decay to the age of the children. Caries attack rates for smooth surfaces could be satisfactorily estimated by this procedure; they seemed on the whole to reflect environmental conditions in the mouth. Rates for pitted surfaces were more difficult to determine because of the tendency of these surfaces to decay rapidly or not at all.

These rates were compared with those for Greek children, and children living in a fluoride endemic area. On pitted surfaces, the rates tended to be similar in all three areas, though differences occurred in the proportion of susceptible teeth. On smooth surfaces, while the rates for this study and for the Greek data were similar, those for the fluoride area were considerably less on both anterior and posterior teeth.

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THE SEXUAL DIAGNOSIS OF ISOLATED BONES OF THE SKELETON

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INTRODUCTION

TO ASCERTAIN the sex of human remains is of great importance both in historical anthropology when studying the typology of a group of people and in certain cases of medico-legal interest.

When the whole skeleton is available, in most cases the skull permits the diagnosis of sex, but when the skull is missing and only isolated bones or parts of bones are available recourse must be taken to the most important sexual differences of such bones to try to effect a diagnosis. Sometimes, even when the skull is available, certain bones of the skeleton permit a diagnosis the correctness of which, if based on the skull alone, would be very doubtful.

In this paper we are interested in examining the probabilities of rendering an accurate diagnosis of the sex of an individual based solely on certain isolated bones of the skeleton independently from the sexual dimorphism of the skull.

The most important sexual differences which a somatoscopic examination shows among various bones have been known for a long time and they are very useful in diagnosing the sex. It is obvious, however, that subjective opinions may influence the diagnosis. If the doubtful cases were few, this would not be a problem, but unfortunately such is not the case.

If quantitative traits of great sexual dimorphism are used, it is evident that all subjectivism disappears. Thus, if a sufficient number of remains of known sex are available, two statistical series can be prepared, one for each sex, and for the diagnosis the midpoint between the averages of the two series could be taken as a basis. Values higher than $\frac{M \male + M \female}{2}$ could be regarded as belonging to one of the sexes, and values lower than that to the other sex. However, as polygons of variability for each sex overlap, it is evident that errors in the diagnosis will be made; the errors will be greater as the overlap between the polygons is greater. On the other hand, if several quantitative traits of sexual dimorphism are known, a discriminant function between them may be calculated, which if applied to the specimens to be studied gives two discriminant series, one for each sex. In general the overlap in variability will be lower and therefore the probability of incurring an error in the diagnosis also will be lower.

For the calculation of discriminant functions it is necessary to have available bones the sex of which is positively known, instead of merely diagnosed. Only in this way will the averages, variability and overlap obtained be really representative of the population being investigated. For the purpose of this paper we have used data relative to individuals of known sex included in the publications of the Anthropological Institute of Coimbra University entitled "Contribuçoes para o Estudo da Antropologia Portuguesa" (Serra, '41, and Tamagnini and Vieira de Campos, '49).

On the other hand, it is realized that the employment of discriminant functions in a specific case will be appropriate only when the specimen under study belongs to the population which has been taken as a basis for the calculation of the function, or to some other population of very similar sexual dimorphism. In fact, this limitation applies with equal force to all methods that may be employed.

If we bear in mind the percentages of probable correctness in sexual diagnosing which according to Hrdlička ('47), may be obtained by adding the skull to other parts of the skeleton, it is believed that the percentages obtained in this paper with isolated bones alone are worthy of consideration. Furthermore, it is not claimed that the calculated functions are final; they only represent a basis for examination which it would be of interest to apply to other measurements of the same bones as well as to other bones of the skeleton. Discriminant functions for the femur and the sternum are calculated.

DISCRIMINATION IN THE FEMUR

The bone material studied for use in the present paper belongs to the "Ferraz de Macedo" collection of the Bocage Museum in the Faculty of Sciences of the University of Lisbon. It is a collection of "identified skeletons" of Portuguese people of about the end of the nineteenth century. Individual data regarding the femora of this series are included in a publication by Tamagnini and Vieira de Campos ('49).

As is well known, the femora, like all other long bones, are usually wider and longer in males than in females. Also, those of the males have more definite muscular insertion reliefs than those of females, but the more important sexual differences are found in the articular extremities which, in the case of males, are generally and relatively greater than in the case of females. Thus, a femur with a large head cannot be a female's, while one with a very small head cannot belong to a male. In intermediate sizes, however, in many instances, the diagnosis is not so easy, if not impossible. This is specifically so in the zone of overlap of the variability polygons of the sexes.

The metrical traits given in the publication by Tamagnini and Vieira de Campos referred to above, which we use here, are: 1) length in standing position; 2) diameter of the head; 3) minimum transverse diameter of the diaphysis; and 4) width of the lower articulation. The techniques of measurement are described in the above-mentioned publication. In the following discussion the traits are numbered 1, 2, 3 and 4, and with them we have calculated, for right and left femora, discriminant functions following the procedure of Fisher ('36 and '49).

In table 1 are shown the respective averages and differences obtained for each trait, and in table 2 are given the *sum of the squares* of the deviation of each variant with respect to the average value (S_{11}, S_{22}, S_{33} and S_{44}), and the *sum of the products* of the deviations of each variant with respect to the average values of each pair of traits (S_{12}, S_{13}, S_{14}, S_{23}, S_{24} and S_{34}).

With the sum of the squares, the sum of the products, and the differences between averages, the following system of equations are propounded:

$$\left.\begin{aligned} S_{11}b_1 + S_{12}b_2 + S_{13}b_3 + S_{14}b_4 &= d_1 \\ S_{21}b_1 + S_{22}b_2 + S_{23}b_3 + S_{24}b_4 &= d_2 \\ S_{31}b_1 + S_{32}b_2 + S_{33}b_3 + S_{34}b_4 &= d_3 \\ S_{41}b_1 + S_{42}b_2 + S_{43}b_3 + S_{44}b_4 &= d_4 \end{aligned}\right\}$$

in which the results of b_1, b_2, b_3 and b_4 are proportional to the coefficients of the discriminant linear function:

$$X = b_1x_1 + b_2x_2 + b_3x_3 + b_4x_4$$

and substituting in that system of equations the values given in tables 1 and 2, the following discriminant linear functions for the right and left femora are obtained:

TABLE 1

Averages and Differences for Each Trait

	Right Femora		Difference	Left Femora		Difference
	Males	Females		Males	Females	
1. Length in standing position	443.95	397.42	46.53	444.69	398.03	46.66
2. Diameter of the head	46.11	39.94	6.17	46.06	39.87	6.19
3. Minimum transverse diameter of the diaphysis	26.13	23.55	2.58	26.44	23.79	2.65
4. Width of the lower articulation	75.60	65.03	10.57	75.44	64.95	10.49

TABLE 2

Sum of the Squares and Sum of the Products

	Right Femora		Totals	Left Femora		Totals
	Males	Females		Males	Females	
S_{11}	26366.84	27215.06	53581.90	27961.04	26903.94	54864.98
S_{22}	314.25	235.78	550.03	337.78	240.95	578.73
S_{33}	248.95	193.44	442.39	253.00	200.27	453.27
S_{44}	871.08	657.94	1529.02	975.00	640.84	1615.84
S_{12}	1553.34	1371.56	2924.90	1698.15	1305.24	3003.39
S_{13}	754.40	994.72	1749.12	708.46	1229.39	1937.85
S_{14}	2427.76	2679.22	5106.98	2708.46	2653.09	5361.55
S_{23}	117.11	119.33	236.44	145.36	140.31	285.67
S_{24}	369.73	272.11	641.84	392.36	267.60	659.96
S_{34}	138.22	154.84	293.06	219.00	198.35	417.35

$$X_R = 0.00019551\,x_1 + 0.00591114\,x_2 - 0.00069119\,x_3 + 0.00391106\,x_4$$

$$X_L = 0.00021523\,x_1 + 0.00661089\,x_2 - 0.00272120\,x_3 + 0.00378059\,x_4$$

in which x_1, x_2, x_3 and x_4 correspond to the four traits under consideration. For easier handling, the coefficient of x_1 may be regarded as equivalent to unity, with the following result:

$$X_R = x_1 + 30.2345\,x_2 - 3.5353\,x_3 + 20.0044\,x_4$$

$$X_L = x_1 + 30.7155\,x_2 - 12.6432\,x_3 + 17.5653\,x_4.$$

If the discriminant function is applied to the differences (d_1, d_2, d_3 and d_4) between averages of the traits under consideration, the difference D between averages of the discriminant functions in males ($\bar{X}$ male) and females ($\bar{X}$ female) is obtained. On both sides we have:

$$D_R = \bar{X}\text{ male}_R - \bar{X}\text{ female}_R = (0.00019551)(46.53) + (0.00591114)(6.17) - (0.00069119)(2.58) + (0.00391106)(10.57) = 0.0851254.$$

$$D_L = \bar{X}\text{ male}_L - \bar{X}\text{ female}_L = (0.00021523)(46.66) + (0.00661089)(6.19) - (0.00272120)(2.65) + (0.00378059)(10.49) = 0.0834112.$$

The analysis of variance may be effected with those values, arranged as follows:

	Sum of squares	Degrees of freedom	Mean Square	Ratio of variance	Probability
Right Femora					
Between groups	$nD^2/2 = 0.2246363$	4	0.0561590	78.511	Very small
Within groups	$D = 0.0851254$	119	0.0007153		
Totals	0.3097617	123			
Left Femora					
Between groups	$nD^2/2 = 0.2156803$	4	0.0539201	76.930	Very small
Within groups	$D = 0.0834112$	119	0.0007009		
Totals	0.2990915	123			

If we take as basis for the discrimination the midpoint between the two averages $\bar{X}$ male and $\bar{X}$ female, there will be error in the classification of individuals who vary more than $D/2$ from the average for their group. The ratio between $D/2$ and the standard deviation of X within the groups permits one to evaluate the proportion of the errors which will be made when applying the discriminant linear function. For the series examined in this paper we have:

$$t_R = \frac{0.0851254/2}{\sqrt{0.0007153}} = 1.5914 \qquad t_L = \frac{0.0834112/2}{\sqrt{0.0007009}} = 1.5753.$$

As we are working with 119 degrees of freedom and, like Mather ('43) we take into account that misclassification happens only in one direction, that is, that only the deviation corresponding to half of the area of the normal curve is of interest, the error becomes reduced to approximately 5% (5.6% and 5.7% of misclassifications for right and left femora respectively).

For appropriate checking we have applied the discriminant functions to each of the individuals included in the series of right and left femora respectively. The averages calculated with the individual discriminant values for the right femora (M male $= 0.6369$; M female $= 0.5518$) and for the left femora (M male $= 0.6138$; M female $= 0.5301$), agree with the theoretical ones previously obtained. Also in agreement are the t values now calculated starting with the differences between the averages (right: M male $-$ M female $= 0.0851$, and left: M male $-$ M female $= 0.0837$) and the sums of the squares (right: Male $= 0.04792$; Female $= 0.03863$, and left: Male $= 0.05004$; Female $= 0.03315$); the degrees of freedom being $n' + n'' - 2 = 122$:

$$t_R = \frac{0.0851/2}{\sqrt{0.08655/122}} = 1.598 \qquad t_L = \frac{0.0837/2}{\sqrt{0.08319/122}} = 1.603.$$

The t values for each trait separately also have been calculated following the same criterion as in the case of the discriminant function:

	1	2	3	4
Right	1.110	1.453	0.679	1.492
Left	1.100	1.417	0.684	1.440

These values are lower than those of the linear discriminant function. It is evident, therefore, that the overlapping variability presented by males and females in each trait becomes smaller when the discriminant functions are used.

It is easy to prove that traits 2 and 4, that is, the diameter of the head and the width of the lower articulation, have t values not much lower than those for discrimination, while, on the other hand, the values of traits 1 and 3 are frankly lower. This calls for some explanation. In the first place we have our calculation for discriminant functions, for right and left femora, using only traits 2 and 4. Following Fisher's method, and without giving here the details of the calculations, the following discriminant functions are obtained:

$$X_R = 0.00617601\, x_2 + 0.00432041\, x_4 = x_2 + 0.699547\, x_4$$

$$X_L = 0.00616322\, x_2 + 0.00397473\, x_4 = x_2 + 0.644911\, x_4.$$

By applying these functions to each of the individuals in both series the following t values are obtained: 1.57 for right femora and 1.55 for left femora. This shows that traits 1 and 3 do not appreciably increase the discrimination. In any case, whenever possible when considering a specific case, it is advisable to use the function of 4 traits as the result will in general be less influenced by chance than if two traits are used, and still much less than if only one trait is used.

DISCRIMINATION IN THE STERNUM

The material studied is part of the excellent collection of "identified skeletons" of the Anthropological Museum and Laboratory of Coimbra University. It consists of 272 sterna of adult Portuguese people metrically and morphologically studied by Serra ('41).

As is the case with most of the bones of the skeleton, the sternum is larger in males than in females. The following traits have been chosen because they afford greater discriminant values: 1) total curvilinear length; 2) total rectilinear length; 3) rectilinear length of the corpus; 4) maximum sub-costal width of the manubrium; and 5) minimum width of the corpus. They are absolute measurements; the indices present small sexual differences in the sternum.

For the calculation of the discriminant function we use now the "size-shape" method of Penrose ('47) because, with 5 traits examined, Fisher's method would be more laborious to employ since for the m traits it requires the calculation of $\frac{1}{2}m(m-1)$ sums of products, m

sums of squares, and the solution of a system of m equations with m unknown quantities.

In table 3 are shown the averages of each trait, expressed in terms of common standard deviation and reduced to a common zero mean.

The "size" value of Penrose, represented by Q, is the result of the sum of the values of the 5 traits in each individual; and the difference Q between males and females is:

$$D_Q = Q \text{ average in males} - Q \text{ average in females} = 9.2220.$$

The "shape" value, repersented by P, is obtained by adding the product of each of the 5 traits by the coefficients k (table 4). The value of the shape difference is:

TABLE 3

Mean Measurements Expressed in Terms of Common Standard Deviation and Reduced to Common Zero Mean

Traits	Males	Females	Difference Male-Female
1. Total curvilinear length	+ 1.1563	— 1.1563	+ 2.3126
2. Total rectilinear length	+ 1.2621	— 1.2621	+ 2.5242
3. Length of the corpus	+ 1.0258	— 1.0258	+ 2.0516
4. Maximum width of the manubrium	+ 0.6248	— 0.6248	+ 1.2496
5. Minimum width of the corpus	+ 0.5420	— 0.5420	+ 1.0840
	+ 4.6110	— 4.6110	+ 9.2220

$$D_P = P \text{ average in males} - P \text{ average in females} = 0.8984.$$

Q and P values, as well as their respective variances and covariances Q/P, were calculated for each individual in both series. By taking for each of these values the average between males and females, and bearing in mind those for D_Q and D_P, the following α is obtained according to the Penrose method:

$$\alpha = \frac{D_Q \text{ Var. } P - D_P \text{ Cov. } QP}{D_P \text{ Var. } Q - D_Q \text{ Cov. } QP} = \frac{(9.2220)(0.6334) - (0.8984)(0.8776)}{(0.8984)(14.5613) - (9.222)(0.8776)}$$

$$= 1.0130$$

and as the best combination of "size" and "shape" is $L = \alpha Q + P$ we have:

$$L = 1.0130Q + P.$$

That function was applied to each of the individuals in both series. Two series were prepared with the individual discriminant values, and the value t was calculated with the difference between the averages (M male — M female $= 10.0605$) and the sums of squares (males $= 2301.06$ and females $= 2400.73$), in accordance with the criterion set down above:

$$t = \frac{10.0605/2}{\sqrt{4701.7952/270}} = 1.21 \text{ (11.3\% of erroneous classifications)}.$$

That is to say, the probabilities of correctness in distinguishing the sex of sterna are 89%.

TABLE 4

	$\bar{♂} - \bar{♀}$	$k = \frac{5(\bar{♂} - \bar{♀}) - 1}{9.2220}$	$\bar{♂} \cdot k$	$\bar{♀} \cdot k$	$\bar{♂} \cdot k - \bar{♀} \cdot k$
1. Total curvilinear length	+2.3126	+0.2538	+0.2935	—0.2935	+0.5870
2. Total rectilinear length	+2.5242	+0.3686	+0.4652	—0.4652	+0.9304
3. Length of the corpus	+2.0516	+0.1123	+0.1155	—0.1155	+0.2310
4. Maximum width of the manubrium	+1.2496	—0.3225	—0.2015	+0.2015	—0.4030
5. Minimum width of the corpus	+1.0840	—0.4123	—0.2235	+0.2235	—0.4470
	+9.2220	—0.0001	+0.4492	—0.4492	+0.8984

SUMMARY

1. The use of discriminant functions, calculated on the basis of quantitative traits, to ascertain the sex of human remains and for the purpose of eliminating the personal judgment in diagnosing, is discussed.

2. For that purpose, data are made available on femora and sterna

of individuals whose sex is positively known, instead of diagnosed, a necessary condition when testing the method of calculation.

3. With Fisher's method, discriminant functions are obtained which permit the sexual distinction of right and left femora with probabilities of accuracy approaching 95%. Likewise, in a series for sterna an accuracy of discrimination of 89% may be obtained with the Penrose method.

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BODY COMPOSITION AND BASAL METABOLISM IN MAN: CORRELATION ANALYSIS VERSUS PHYSIOLOGICAL APPROACH [1]

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WHILE the relation between basal metabolic rate (BMR) and body size has been examined in numerous studies (cf. Kleiber, '47), there is much less information on BMR and body composition, in the chemical and anatomical sense. Basal metabolic rate represents the sum of energy metabolism of all the cells of the body under standard conditions. Since there are in the body various kinds of cells, differing in the rate of oxygen consumption, it is clear that BMR must be related to the proportion of the different cells or tissues constituting the particular body.

Several attempts have been made to "partition" the total basal oxygen consumption of the animal body among its different organs or tissues but progress in the solution of the problem, clearly stated by Barcroft as far back as 1908, has been slow. In the last few years, however, the development of methods for determining the composition of the human body *in vivo* has given a new impetus to this type of studies. The correlations between different components or compartments of the body and BMR were investigated by several authors. Such studies are important both from a theoretical and a practical point of view. A better knowledge of these relationships is essential for the understanding of the physiological factors which govern the magnitude of the BMR. Also, it will provide a more adequate basis for establishing BMR norms, used for the comparison of the different individuals and the evaluating of the deviations from "normality," than can be obtained from the height and gross body weight alone.

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Two main approaches have been followed in the studies of the relationship between body composition and BMR. On the one hand, it has been possible to measure directly, by physiological techniques, the oxygen uptake of various organs of the human body, *in vivo,* and to evaluate their share in the basal oxygen consumption of the intact body. We will refer to this method as "physiological approach." On the other hand, the study has been pursued by applying the methods of statistical analysis and calculating the coefficients of correlation between the magnitude of some compartments of the body and the BMR. We shall speak in this case of "statistical approach." Unfortunately, the evaluation of the latter data has not always been carried out with the necessary care and caution, and in some cases the interpretation of the correlational analysis conflicts with the results obtained by the direct methods (the "physiological approach").

Modern statistical methods provide useful tools for the design of biological and medical investigations and for the scientific analysis of the data. At the same time, the increasing complexity of statistical operations magnifies the danger of an incorrect biological interpretation of the correctly computed statistics. This danger is present even when elementary statistical concepts and procedures, such as correlational analysis, are used.

In correlational analysis, as applied to the study of basal metabolism and body composition, the *association* between specific criteria of body composition and basal metabolism has been interpreted by some authors incorrectly in terms of *causation.* While valid information obtained by statistical analysis of data on total oxygen intake and a body compartment is limited to individual *differences* (interindividual variation) and the *covariation* of basal metabolism with morphological characteristics, erroneous inferences have been made on the *partitioning* of basal oxygen consumption among different body compartments. Such incorrect conclusions, based on careful measurements and correctly ascertained statistical relationships, have crept into a recent paper by Garn, Clark and Portray ('53).

The authors stated clearly (p. 166): "Muscle and fat together account for the major proportion of the child's oxygen needs under basal conditions." In summarizing their findings they wrote (*loc. cit.*): "Together fat and muscle accounted for over 70% of the basal oxygen consumption." This conclusion was based on the fact that the coefficients of correlation between BMR and the calf muscle measurements (maximum antero-posterior diameter) were 0.82 for boys and 0.73 for girls; for "fat" (difference between total diameter and the muscle diameter), the values were 0.30 and 0.35, respectively; no coefficients of multiple correlation were reported in which "fat" and "muscle" were combined.

With the variance of the basal consumption values taken as unity, one must beware of simply adding the proportions accounted for by each of the several criteria of body structure (such as skinfolds, limb circumferences, or bony dimensions). By such a procedure one could readily exceed unity. It is the coefficient of multiple correlation, squared, which must be used in determining the amount of variation in one measurement, such as basal metabolism, accounted for on the basis of several other measurements considered simultaneously. Even this procedure remains limited to "external," *statistical associations* which say or imply nothing about "internal," *physiological dependence*.

In view of the current interest in the analysis of the relationships between body composition and BMR, it seems desirable to attempt to clarify the essential differences between the correlational analysis and the direct estimations of oxygen consumption. It is hoped that a critical examination of these two approaches to the problem will be helpful in correctly interpreting available data and will encourage the integration of fundamental information obtained by different methods.

CORRELATIONAL ANALYSIS

Because of the difficulty of a direct, "intuitive" interpretation of a coefficient of correlation, the r is perhaps the least useful way of expressing the association between two variables. For purposes of prediction, the relationship is expressed in the form of a regression equation of the type $Y = a + bX$, togethed with the standard error of estimate $(SEE_Y = SD_Y\sqrt{1 - r^2_{XY}})$ which serves as an absolute criterion of how "good" is the correlation between Y (basal metabolism) and X (a measure of body composition).

When one wishes to indicate how much of the total variation in basal metabolism is accounted for by the regression, it is necessary to use the coefficient of determination, r^2. This can be written simply as $r^2 = (SD_{\hat{Y}})^2/(SD_Y)^2$. The ratio, then, indicates the fraction of the variance of the Y values which is statistically accounted for on the basis of the association of Y with X. It is in this sense that Garn and his co-workers wrote "Together these two tissues, muscle and fat, appear to account for at least 70% of the total interpersonal variance in the basal metabolic rate in the age group considered."

However, the argumentation was switched from a *statistical* into *physiological* accounting, from the analysis of covariation between body structure and basal metabolism to the partitioning of basal oxygen consumption. The fraction of interindividual variance in oxygen intake, accounted for statistically on the basis of correlations with the linear calf dimensions and indicated by r^2, can not be identified with the fraction of the total basal oxygen consumption represented by the energy metabolism of skeletal muscles and the adipose tissue.

Researches on tissue and organ respiration indicate that the partition of the basal oxygen consumption is strikingly different from that inferred from the correlational analysis. On the basis of our present knowledge, muscle and adipose tissue will in no case account for 70% of the total energy metabolism of the human body under standard resting condition.

PHYSIOLOGICAL PARTITIONING OF BASAL OXYGEN CONSUMPTION

Information is available on the oxygen consumption of many of the organs of the human body under resting conditions. This makes possible approximate partitioning of the total oxygen consumption of the resting man. A calculation of the relative participation of the different organs in the total oxygen consumption was made some time ago by one of us (Grande, '50) and by Drabkin ('50). Since additional data have been published in the last few years, it may be appropriate to reevaluate the share of the different tissues and organs in man's basal oxygen consumption. In order to make such estimations we have to know the rates of oxygen consumption in different tissues and the mass of these tissues.

To begin with the single largest anatomical component, the amount of muscle tissue in the normal adult man is generally estimated to be about 40% of the total body weight. In table 1 two values are cited from the classical German literature, together with recent measurements made by American authors. The average amount of muscle in the "normal" adult man is not over 40% of the total body weight and is probably less than that.

As to the amount of oxygen consumed by resting human muscle, the figure of 0.17 cc O_2 per 100 gm per minute was obtained by Asmussen, Christensen, and Nielsen ('39). The recent direct determinations made by Mottram ('54) yield an average value of 0.245 cc per 100 cc muscle per minute, with a standard deviation of 0.07 cc. This value, corrected for the density of muscle, gives a value of 0.23 cc O_2 per 100 gm of muscle per minute. By applying these figures to the values of muscle mass it is possible to estimate the oxygen consumption of the total musculature of a "normal" man. A minimal value of some 37.4 cc per minute and a maximal value of 69.2 cc is obtained. Taking 250 cc O_2 per minute as the value of the basal metabolic rate of a normal adult man, the above figures would correspond to 15% and 28%, respectively, of the total oxygen used. It is clear, therefore, that even using the highest value the musculature could not contribute more than some 30% of the total basal oxygen consumption.

TABLE 1

Measured amounts of skeletal muscle tissues in the human body

Author	% of Total Body Weight	Kg in a 70 Kg Man
Theile (Suárez, '52)	35.9	25.1
Vierordt ('06)	43.0	30.1
Mitchell, *et al.* ('45)	31.5	22.0
Forbes, *et al.* ('53)	39.7	27.8

A comparison of the O_2 consumption of the musculature with that of some important organs may be instructive. In table 2 the values were calculated for the liver, brain, heart and kidneys as well as for the musculature, using for total muscle the value of 39.7% of the total body weight found by Forbes *et al.* ('53), and the values for oxygen consumption of the different organs given in the literature.

From the foregoing calculations it can be concluded that the internal organs account for over 60% of the total basal oxygen consumption. The remaining organs and tissue other than muscles (skin, bones and bone marrow, lungs, adipose tissue, etc.) undoubtedly have a certain, though small, consumption of oxygen. In view of the existing experimental evidence it is impossible to accept an oxygen consumption for the human resting musculature much higher than some 25% of the basal oxygen consumption of the total body.

As to the oxygen consumption of the adipose tissue, there is general agreement that it has a very low energy metabolism and the calculations of table 2 indicate that this cannot be greater than a fairly small fraction of the 13% to 14% of the basal oxygen consumption not accounted for by the metabolism of liver, brain, heart, kidney and skeletal muscle.

TABLE 2

Physiological partitioning of basal oxygen consumption in the normal adult man (body weight, 70 kg; body surface, 1.73 m^2; resting oxygen consumption, 250 cc O_2 per minute).

Organ	Weight, gm	Weight, % total body	Oxygen consumption, cc/100 gm/minute	Total O_2 consumption	% of BMR
1. Liver (a)	1,500	2.1	4.4	66	26.4
2. Brain (b)	1,400	2.0	3.3	46	18.3
3. Heart (c)	300	0.43	9.4	23	9.2
4. Kidneys (d)	300	0.43	6.1	18	7.2
			Subtotal (1-4)	153	61.1
5. Skeletal muscles (e)	27,800	39.7	0.23	64	25.6
			Total (1-5)	217	86.7

(a) Includes total oxygen consumption of "splanchnic area." *Cf.* Bradley *et al.* '45; Myers, '47; Myers and Holland, '49; Dobson *et al.*, '53.

(b) Kety and Schmidt, '48; Scheinberg and Stead, '49; Heyman *et al.*, '50; Stead, '50; Bentinck *et al.*, '51; Schieve *et al.*, '51; Novak *et al.*, '53.

(c) Calculated assuming left ventricle = ⅔ of total heart weight. The oxygen consumption rate of the right ventricle is assumed to be ½ of the left ventricle. *Cf.* Bing *et al.*, '49, '50.

(d) Cf. Bradley and Halperin, '47; Clark and Barker, '49; Smith, '51.

(e) Calculated with Mottram's ('54) figure corrected for muscle density.

COMMENT

In recent years a good deal of progress has been made in the partitioning of the body, *in vivo*, using external body measurements, including skinfolds, X-rays, body density, and body water and its fractions (for a summary cf. Brožek, '53; Keys and Brožek, '53). This provides new tools and a new frame of reference for the analysis of biochemical processes and physiological functions which are related to the composition of the body.

It has been noted that the basal oxygen consumption of different tissues and organs varies markedly. Consequently, the separation of gross body weight into components which have no or low energy metabolism (extracellular water, bone mineral, fat) and those which are characterized by substantially higher oxygen requirements appears useful. Theoretically, at least, a better reference point for the evaluation of normality of the basal oxygen consumption is provided by such separation than by using gross body weight or other derived values, such as the surface area calculated from weight and height. The significance of changes in basal metabolism observed in prolonged caloric deprivation (Keys *et al.*, '50) or in the course of aging (Brožek, '52) may be interpreted physiologically more correctly when these changes are referred to the "fat-free body mass" or "active tissues" (the latter being defined as body weight minus the weight of fat, bone mineral, and extracellular fluid) than to gross body weight.

"Active tissue mass" represents the sum of bodily tissues which participate more actively in the resting energy metabolism of the body. The concept is useful and progress in the estimation of different body compartments has made possible a more objective characterization of its boundaries.

However, the physiological partition of the basal oxygen consumption of the human body points out some important limitations which should be borne in mind when speaking of "active tissue" mass. It is not an homogeneous mass of tissues all having an identical metabolic rate. The figures given in table 2 clearly indicate that the "active mass" of the body is in fact made up of a relatively small mass of visceral organs (liver, brain, kidneys, heart, etc.) with a relatively high metabolism under resting condition of the body, and a much greater mass with a relatively low resting metabolic rate, constituted by the skeletal musculature. Obviously, the "active tissue mass" can not be identified with the skeletal musculature.

The erroneous idea that the skeletal muscles have a large share in the total oxygen consumption of the body at rest has a long tradition in physiological thinking. In his classical review of 1908 Barcroft criticized the idea put forward by the French physiologist Richet, according to which the musculature makes up over 90% of the total oxygen consumption of the resting body. This idea seems to persist in the minds of present-day physiologists, who, in speaking of active tissue mass or "metabolically active mass," tend to identify it with the skeletal musculature.

The resting metabolic rate of the human body is no doubt a measure of the "active mass," but, as this mass is made up of different components with different metabolic rates, it is impossible to translate the BMR figures directly into actual weight of the "active tissue mass." Obviously, a change in the proportions of the components of the "active mass" will produce changes in the basal metabolic rate, without a change in the total mass.

It has been stressed earlier that a statistically significant correlation between two variables does not prove any direct causal relationship in a physiological sense. The association may be indirect and result from the correlation of both variables with other variables. The interpretation of a coefficient of determination in terms of the share of a body component in the basal metabolism can be reduced *ad absurdum* by considering the high correlation between the fluid compartments of the body and the basal oxygen consumption. Thus Wedgwood *et al.*, ('53) reported an r of 0.80 between interstitial fluid and basal metabolic rate. While this fluid compartment "accounts" statistically for 64% of the variation in the BMR, its actual participation in the body's oxygen consumption must be practically zero. No direct causal relationship is present at all, in spite of the fairly close parallelism between the two variables.

SUMMARY AND CONCLUSIONS

Statistical "accounting" for a quantitative phenomenon, such as basal metabolism, consists in comparing the variance of the values ($\hat{Y}$), predicted on the basis of the criterion (X), with the variance of the dependent variable (Y).

With X representing a muscle diameter (or a more direct measure of muscle mass) and Y representing the basal metabolism, the coefficient of determination (r^2) cannot be interpreted as the relative share of the muscle mass in basal oxygen consumption.

Direct determinations of the oxygen consumption of human muscles, based on the rate of blood flow and the arterio-venous oxygen difference, suggest that in the resting state the skeletal musculature is responsible for only about 25% of the amount of O_2 consumed by the body.

The separation of the body into components which have a low or no energy metabolism (fat, extracellular fluid, bone mineral) and those which are characterized by a substantially higher oxygen intake is useful. However, it should be kept in mind that the "active tissue mass," obtained as the difference between body weight and the sum of the weights of the "inactive" compartments, is heterogeneous in composition and in the rate of oxygen uptake of its components.

The "active tissue mass," under resting conditions, can not be identified with the muscle mass.

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VENEREAL DISEASE CONTACTS OF A SPECIFIC GROUP OF MEN IN A 5-DAY PERIOD

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VENEREAL disease continues to be one of the major problems for all medical officers in the Armed Forces, especially in overseas areas. The incidence of such disease is directly related to three factors, namely, incidence of exposure, effectiveness of available prophylaxis, and how such prophylaxis is used. The purpose of this paper is to report the incidence of exposure of a given group of enlisted men during a 5-day period.

Prior to the work of Kinsey and associates ('48), little statistical information was available as to prevailing sexual activities of men. Most of the information was based on individual case histories primarily from the psychiatric literature, "clinical impression," and personal experience. Owing to different educational, social and economic backgrounds and the fading of youth, many line officers and medical officers are unaware of the extent of sexual activities of young enlisted men. It is hoped the present report will supply additional information on the incidence of exposure for those concerned with the control of venereal disease.

In 1949, the author was attached to a group serving in the Far East. For two months the enlisted men concerned had been in a cold climate with little in the way of liberty or recreation. Opportunities had been available and a few of the less esthetically inclined had contracted various venereal diseases, but the over-all incidence was about the same as for all military units in the area. Then, after the group spent 5 days in one of the more modern cities of Asia in a warm climate, with liberty being granted to two thirds of the crew from 1 p. m. to midnight each day, more venereal infection developed in a few days than had been encountered in the preceding few months. Prior to this liberty extensive lectures on venereal disease and prophylaxis had been given, educational movies had been shown, and all available types of prophylactics had been provided.

A questionnaire was prepared for the purpose of finding out which one of the three variables mentioned in the first paragraph was responsible for this great increase in venereal disease. The questionnaire was passed out 4 days after the liberty period had ended and while the men were at sea returning to their usual duty bases. With the men at quarters, it was announced over the public address system that the medical department was greatly concerned over the sudden increase in venereal disease, that the co-operation of the men was needed to plan more effective treatment for the future, that the information was to be used only by the medical department and that the questionnaires would remain anonymous. The questionnaires and pencils were distributed and rapidly collected, the entire time, including that used for the announcement, not exceeding 10 or 15 minutes. The questionnaire included the following items:

1. Age.
2. Married or single.
3. Is this your first or second trip to the Far East (or more)?
4. How many times did you go on liberty in [the designated city]?
5. Do you drink alcoholic beverages?
6. How many sexual contacts did you have in [the designated city]?
7. How many sexual contacts were made after drinking?
8. Would you have made these sexual contacts if you had not been drinking?
9. How many times did you use only a rubber condom?
10. How many times did you use only a Navy sanitube?
11. How many times did you use both a condom and a sanitube?

Approximately 200 of the questionnaires were distributed and 188 were returned. Of these, 5 represented men who had not been on liberty, and 11 were improperly filled out (interestingly enough, only one of these facetiously); hence, there remained 172 questionnaires which form the basis of this report. Of the 172 men, 126 were single, 45 were married and one was divorced.

In regard to the number of trips to the Far East, 83 single men were on their first, 36 were on their second and 7 were on at least their third trip. The corresponding figures for the married men were 26 on the first trip, 14 on the second and 5 on at least the third trip. Combining the figures, one finds that 64% of all the men were on their first trip, 29% were on their second trip, and 7% were on at least their third trip. The single men had a total of 366 liberties, the married men had 131 liberties and the one divorced man had 4 liberties. This makes a total of 501 liberties by the entire group during the 5 days for an average of 2.9 liberties per man. There was no significant difference in the number of liberties the single men had as compared to the married men—they all went on liberty as often as they could.

TABLE 1

Contacts Made in the 5-day Period by the Given Number of Men, Tabulated According to Age and Marital Status

Age	Marital status	Contacts 0	1	2	3	4	5	6	7	8	9	10	11	12	16	Total
17	Single	2	4													6
	Married															
18	Single	6	10	6	1	2	1		1		1					28
	Married			1												1
19	Single	6	9	2			1							1		19
	Married		1													1
20	Single	5	5	4	4	3	1			2	2			1		27
	Divorced											1				1
	Married			1	1											2
21	Single	4	3	2	3	1	4							1		18
	Married	2			1											3
22	Single	5	1				1	1								8
	Married			3	1											4
23	Single	2	1	3				1								7
	Married	3		1												4
24	Single		2	1			1	1								5
	Married	1	1	1	1	1										5
25	Single			1												1
	Married	1		1	1											3
26	Single	1														1
	Married				1	1							1			3
27	Single				1										1	2
	Married	3		2												5
28+	Single	2		1		1										4
	Married	9	2	1	2											14
Total		52	39	31	17	9	9	3	1	2	3	1	1	3	1	172

Table 1 shows the number of contacts made by the men during the 5-day period. (For the purposes of this study, a contact is defined as an act of intercourse.) For example, of the 17-year-old single men, two had no contacts and 4 had one contact each. One may summarize this information by saying that the entire group had a total of 374 contacts. These were made by 120 men for an average of 3.1 contacts per man. On a percentage basis, 70% had contacts. The 93 single men had a total of 295 contacts, which means that 74% of the single men had contacts and averaged 3.2 contacts per man. The 26 married men had a total of 69 contacts, which means that 58% of the married men had contacts and averaged 2.7 contacts per man.

TABLE 2

Relation of Use of Alcohol to Number of Contacts

	Do you drink alcohol?		Would you have had contacts if not drinking?		Contacts made after drinking									
Age	Yes	No	Yes	No	1	2	3	4	5	6	7	8	11	12
17	6		3	1										
18	24	4	20	3	10	2		2			1	1		
19	19	2	14		6	1								
20	27	2	20	4	5	6	3	3		1		1		
21	17	4	14	1	2	3	3	1	2					
22	11	2	4	3	3	1		1						
23	10	1	5	1	1	3				1				
24	10		9		4	2	1			1				
25	4		3			2	1							
26	4		2				1	1					1	1
27	5	2	4				1							
28+	15	3	5	3	2	3								
Total	152	20	103	16	33	23	10	8	2	3	1	2	1	1

Alcohol is undoubtedly a factor in the transmission of venereal disease. Shakespeare noted that alcohol "Provoketh the desire. . . ." In the present study the author tended to blame the cheap and abundant alcohol for the great amount of sexual activity during the 5 days of liberty. Most of the information obtained from the questionnaire in regard to the use of alcohol is shown in table 2.

The information about alcohol can be summarized as follows: one hundred and fifty-two men or 88% stated that they used alcohol, there being little difference here between the married and single men. Of the 374 sexual contacts, 215 or 57% were made after drinking. There was found to be some difference here between the married and the single groups, as 81% of the contacts by married men were made after drinking while only 53% of the contacts made by the single men were after drinking.

The question "Would you have had these contacts if you had not been drinking?" was used as a check on the reliability of the answers. It was felt that if a man answered "yes" to this question, then most of his other answers probably would be reliable. On the other hand, if he were seeking an "out" or justification for his actions and did drink, he would have been inclined to blame the alcohol and answer this question "no." Of those answering the question, 103 out of 119 did so in the affirmative. On a marital basis, 8 of 93 single men and 8 of 26 married men blamed the alcohol.

Because the types and methods of prophylaxis used by the Armed Forces have changed considerably since this study was made in 1949, no detailed analysis of this information will be given here. It suffices to say that of the 120 persons having contacts, only 26 failed to use any protection at all. They had 91 unprotected contacts or 24% of the total made by the group.

The next step in this study was to see if there was any significant difference between the behavior of this group of 172 men who, for the most part, did not contract a venereal infection and another group of men who did. The latter group consisted of 49 patients. These were obtained from 4 similar groups of men who had spent 5 days in the city and to whose records the author had access, 10 of these patients being from the original group of some 200 men on which the major part of this paper is based. Of these 49 patients, the author was able to interview 39 and gather data on behavior. Hence, these 39 men make up the venereal disease group on which the following comments are based.

Eight of the 39 venereal disease patients were married. The average number of contacts, 2.3 per man, was the same for married and single men. This rate is actually somewhat less than those for the control group of 172 men (2.7 for the married and 3.2 for the single men), although it is doubtful that the difference is significant. The group of men contracting an infection was essentially similar to the control group in age, number of liberties, trips to the Far East, and so forth. The only striking difference was that 96% of the contacts by the group who contracted an infection were made after drinking as compared to

57% for the control group. Moreover, the group who contracted an infection used some form of prophylaxis for only 45% of their contacts as compared with 76% for the control group. Thus alcohol does not seem to play much of a role in determining whether or not the men will have sexual contacts, but those who do drink use less precaution and are more likely to contract an infection than are those who do not drink.

During the time the author was interviewing the men who had contracted an infection the study was reviewed by the area consultant in psychiatry. At his suggestion and in the hope of finding further information as to why these men happened to contract an infection, histories of sexual development were taken where possible. The results are given in table 3. It is of interest to note that 12 of 15 men had experienced intercourse before they reached the minimal military age of 17 years. This suggests that their behavior patterns were well established before they entered military service, and hence it seems to contradict the charges sometimes made that the Armed Forces corrupt the morals of men.

These 39 cases are too few to permit the drawing of any conclusions as to the type of person who contracts a venereal infection. The frankness of some of the answers, however, would tend to indicate that the data are probably reliable.

SUMMARY AND CONCLUSIONS

This is a report primarily on the sexual contacts of a group of 172 enlisted men. While some of the figures may seem unlikely, all are within the ranges for sexual activity as listed by Kinsey and co-workers. During 5 days of liberty, 70% of the men had contacts. While 88% of the men stated that they used alcohol, only 57% of the contacts were made after drinking. Of those who answered the specific question, more than 80% said they would have had the contacts whether they had been drinking or not. The behavior of this group has been compared with that of a smaller group of men who did contract venereal infections. The main difference seems to be that the group contracting infections more often used alcohol before and less often used protection during contacts than did those who escaped infection.

The author wishes to emphasize that these data represent the action of a given group of men under a given set of circumstances. No conclusions are drawn as to whether this reported behavior is normal or abnormal, as to whether it is representative of young men in the Armed Forces, or as to whether it is even representative of the usual behavior of this group of men.

TABLE 3

Data on Sexual Development

Case	Age	Contacts in city	Education (years)	Age first masturbated	Age first intercourse	Age or time last masturbated	Approx. range of contact per week in States (before Far East)	Married or single
A–W–1	17	1 for 1	11	11	15	15	4–12	S
B–H–10	19	3 for 12	12	11	17	18	3–4	S
B–H–11	21	6 for 10	12	12	15	16	2–3	S
B–H–12	17	2 for 4	9	11	17	2 wk. ago	1–2	S
B–H–13	20	2 for 20	9	12	15	15	9–12	M
B–H–14	27	2 for 7	12	11	14	15	1–2	M
B–H–15	22	2 for 3	7				2–3	S
B–H–16	20	3 for 10	10	12	16	2 wk. ago	1–2	S
C–B–1	22	1 for 1	11	11	15	7 mo. ago	1–2	S
C–B–2	18	3 for 3	12	12	17	1 mo. ago	1–2	S
C–B–3	21	2 for 2		12	16	17	1–2	S
D–T–4	18	3 for 13	10	11	13	15	< 1/wk.	S
D–T–5	19	1 for 10	12	11	14	3 mo. ago	< 1/wk.	S
D–T–6	18	3 for 10	8	14	14	1 mo. ago	3–4	S
D–T–8	18	1 for 2	11	12	16	10 mo. ago	3–4	S
D–T–9	21	1 for 7	11	12	14	3 mo. ago	4–6	S

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FAT ACCUMULATION AND WEIGHT GAIN IN THE ADULT MALE

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INTRODUCTION

BY THE third decade of life the male has reached maturity, a life-phase marked by a cessation of vertical growth. The termination of skeletal increases, however, and an abrupt end to increments in "operational" tissue often herald a gain in fat-containing tissue, and progressive alterations in the surface contours.

These changes in the deposition and accumulation of fat, though unaesthetic and possibly linked to later degenerative disorders, are of particular interest to the student of growth and form. For the thickness of the subcutaneous fat layer is accessible to measurement, and the analysis of simultaneous changes at different parts of the body provides insight into one aspect of changing body form.

At the same time studies dealing with the deposition of fat are valuable in comprehending the meaning of "normal" weight, and the significance of weight changes. It is not how heavy a man is or how heavy he becomes that has maximum prognostic significance, but rather how much fat he carries and how much fat he adds. It is not unlikely that the location of the fat, as well as the gross amount, may elucidate the differential mortality rates of the different weight groups.

Without reviewing the growing bibliography on body composition (see Brožek, '53; Reynolds, '51) it is sufficient to say that only limited information is available on the thickness of uncompressed subcutaneous fat-containing tissue in adult males (Garn, '54) and that fewer data have been published on the differential increase in subcutaneous fat with advancing age and weight. Moreover the possibility of using such data in the analysis of form has not been a stated aim of most published research in this field.

It is the purpose of this communication, then, to report on age-associated changes in 9 measured thicknesses of the subcutaneous fat in adult males, and to express these changes in relation to measured weight and the estimated weight of fat. It is a further purpose to express these fat-thickness and fat-weight relationships in a manner that may be helpful in comprehending how the superficial fat-containing layer alters its configurations as the totality of fat becomes greater or less.

METHODS AND MATERIALS

Roentgenographic and other studies were made on a total of 85 white males from 20 to 69 years of age. All were active and reasonably healthy and were for the most part parents of participants in the Fels longitudinal growth investigations. Occupationally the group included farmers, professional and semi-professional workers. In terms of national origins, all except two were of Northwest European ancestry. One extremely obese male weighing 140 kg and one male with multiple congenital defects were not included in the tabulations.

Besides routine measurements of height (standing) and weight (nude), a total of 9 fat-shadow measurements were made on standardized soft-tissue teleoroentgenograms, using a micrometer caliper with ground tips (Garn and Saalberg, '53). The fat shadow measurements included deltoid-insertion fat, lateral arm fat, medial arm fat, iliac fat, trochanteric fat, posterior leg fat, anterior leg fat, medial leg fat and lateral leg fat. The location of the sections X-rayed and measured as well as the poses adopted are shown in Garn ('54).

The creatinine values used in this study are based on full 24-hour urine collections, acidified and refrigerated until analysed. Determinations were made as described by Clark and Thompson ('49) using the Beckman spectrophotometer and a creatinine standard. All values were expressed as creatinine (in milligrams) per 24 hours.

The estimated weight of fat, as reported here, was based on the trochanteric fat thickness, since it has been shown that trochanteric fat is the best single indicator of subcutaneous fat thickness in general and of the 9 thicknesses measured, it is best correlated with weight (Garn, '54). The weight of fat, corresponding to a given trochanteric fat thickness, was computed from the correlation coefficient between trochanteric fat and weight, and the standard deviations of weight and trochanteric fat respectively. It may be thought of as the weight that would have to be deducted from the subject's observed weight, if he were reduced to a trochanteric fat thickness of 0.[1]

FINDINGS

As shown in table 1, 8 of the 9 fat-containing layers measured exhibited an age-associated increase, with the maximum gain both absolute (7.8 mm) and relative (58%) attained by the iliac fat. The three thickest layers, iliac, trochanteric and deltoid-insertion fat all increased 3 mm or more. The 6 distal fat layers, thinner to begin with showed smaller absolute gains, and smaller percentage increases. Notably, the rate of increase of iliac fat out-paced that of the other fat layers, so that by the fifth decade iliac fat was the thickest, reversing its earlier ranking. The data do show that there is a tendency to deposit fat differentially over the pelvic region. However it is evident that the rate of increase differs even on different quadrants of the lower leg, and that the deltoid-insertion fat falls between the "central" and the "distal" fat both anatomically and in its pattern of gain.

Over the 4 decades considered here, weight also rose, paralleling the increase in fat. In weight, as in fat thickness, the change was more rapid between the third and fourth decades. The curvilinear nature of these changes made η rather than r the appropriate measure of relationship with age. As shown in table 1, the etas approach or exceed significance at the 5% level, though it is acknowledged that the meaning of a given value of η is open to question (Fisher, '48, p. 256).

Since the estimated weight of fat is derived from the trochanteric fat thicknesses, this later value parallels the increase in trochanteric fat, rising from 11.5 kg in the third decade, to 17.3 kg in the sixth. If computed as a percentage of gross body weight, the change is from 15.6% to 22%, a not unreasonable value when compared to other studies. The same data computed upon an individual basis suggest a lean body weight (gross weight minus estimated fat weight) of 61.5 kg in the third decade, 60.4 kg in the fourth decade, 61.4 kg in the fifth, and 58.9 kg in the sixth. The creatinine values, obtained on 30 of the subjects over 30 years of age show a negative relationship with age, with the regression equation being Creatinine = —7.56 Age + 2112. It should be noted, however, that the stature of the oldest age group (175.3 cm) is also the lowest by nearly 2 cm so that a possible decrease in muscle mass, as suggested by the creatinine trend, may be attributed to smaller size, rather than to an actual loss of muscle.

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[1] In computing the estimated weight of fat, from the regression of weight on trochanteric fat, and in computing the estimated fat-free weight (observed weight minus estimated weight of fat) no corrections for stature were made since the mean stature was substantially the same for all 4 age-groups.

TABLE 1

Age changes in fat thicknesses, weight and estimated fat weight

Measurement		Age group				Relationship with age (η)
		20–29	30–39	40–49	50–	
No. of men		15	18	32	20	
Age	(yrs)	24	34	44	55	
1. Deltoid insertion fat	(mm)	14.8 ± 1.3 [1]	18.6 ± 1.1	18.7 ± 0.6	18.3 ± 0.8	0.31
2. Lateral arm fat	(mm)	4.2 ± 0.5	4.7 ± 0.4	4.3 ± 0.3	5.1 ± 0.4	0.21
3. Medial arm fat	(mm)	3.2 ± 0.3	3.3 ± 0.3	3.4 ± 0.2	4.1 ± 0.3	0.25
4. Iliac fat	(mm)	13.5 ± 1.9	17.9 ± 2.0	20.8 ± 1.3	21.3 ± 2.2	0.34
5. Trochanteric fat	(mm)	12.3 ± 1.9	16.6 ± 1.4	17.5 ± 1.2	18.4 ± 1.5	0.30
6. Posterior leg fat	(mm)	6.5 ± 0.7	7.3 ± 0.6	6.9 ± 0.4	8.2 ± 0.4	0.23
7. Anterior leg fat	(mm)	2.8 ± 0.2	2.8 ± 0.1	2.6 ± 0.1	2.8 ± 0.2	0.04 [2]
8. Medial leg fat	(mm)	5.0 ± 0.5	6.3 ± 0.6	6.2 ± 0.4	7.3 ± 0.7	0.30
9. Lateral leg fat	(mm)	4.1 ± 0.4	5.3 ± 0.4	4.5 ± 0.3	4.7 ± 0.4	0.24
10. Estimated weight of fat	(kg)	11.5 ± 1.8	15.6 ± 1.3	16.3 ± 1.1	17.3 ± 1.4	0.31
11. Weight	(kg)	73.3 ± 2.5	76.3 ± 1.9	78.1 ± 1.6	77.1 ± 1.9	0.18 [2]
12. Height	(cm)	176.5 ± 1.0	177.4 ± 1.8	177.3 ± 1.0	175.3 ± 1.9	—

[1] Mean ± s. e.

[2] Not significant.

Inasmuch as the age "changes" detailed here are primarily changes in fat-containing tissue and weight, they are best illustrated by showing the relationships between the estimated weight of fat, and the 9 subcutaneous fat thicknesses (table 2). As also shown in figure 1, all of the fat thicknesses rise with the estimated weight of fat, including the anterior leg fat, a trend which has been verified separately. Iliac, trochanteric and deltoid-insertion fat form one cluster of lines while the distal fat thicknesses form a second cluster. The "crossing over" of iliac and deltoid-insertion fat is clearly shown. What is especially evident in figure 1 is the fact that the accumulation of fat with age follows the general fat-weight, fat-thickness trends, and that older men of average weight for age have the surface contours that would be expected for younger men 0.5 standard deviation or 5 kg (7%) "overweight" for their age.

TABLE 2

Relationship between fat and weight changes

Measurement	Mean thickness (mm)	Weight change (kg) per mm fat[1]	Fat change[2] (mm) per kg weight
1. Deltoid insertion fat	17.8	1.35	0.74
2. Lateral arm fat	4.6	3.32	0.30
3. Medial arm fat	3.5	3.15	0.32
4. Iliac fat	18.9	0.69	1.45
5. Trochanteric fat	16.6	0.94	1.06
6. Posterior leg fat	7.2	1.49	0.67
7. Anterior leg fat	2.7	7.93	0.13
8. Medial leg fat	6.3	2.69	0.37
9. Lateral leg fat	4.7	1.64	0.61

[1] Calculated from formula $y = r\frac{\sigma y}{\sigma x}x$ using values given in Garn ('54). The actual age-associated changes in fat closely follow the predicted values, as shown in figure 1.

[2] Calculated from column 2.

Under these circumstances, then, "ageing" in the male, within the limits set by this study and the age span considered, closely corresponds to ordinary fattening. The surface contours of the average older man bear a marked resemblance to the surface contours of the fatter younger man, being greatest about the hips, at the trochanteric region, and on the upper arm. Less evident to the eye, but equally important, fat is added peripherally, but at a lesser rate, and to layers initially thinner.

DISCUSSION

It is evident, from the preceding findings, that ageing in the male, or more precisely, the simple passage of time, is accompanied by an

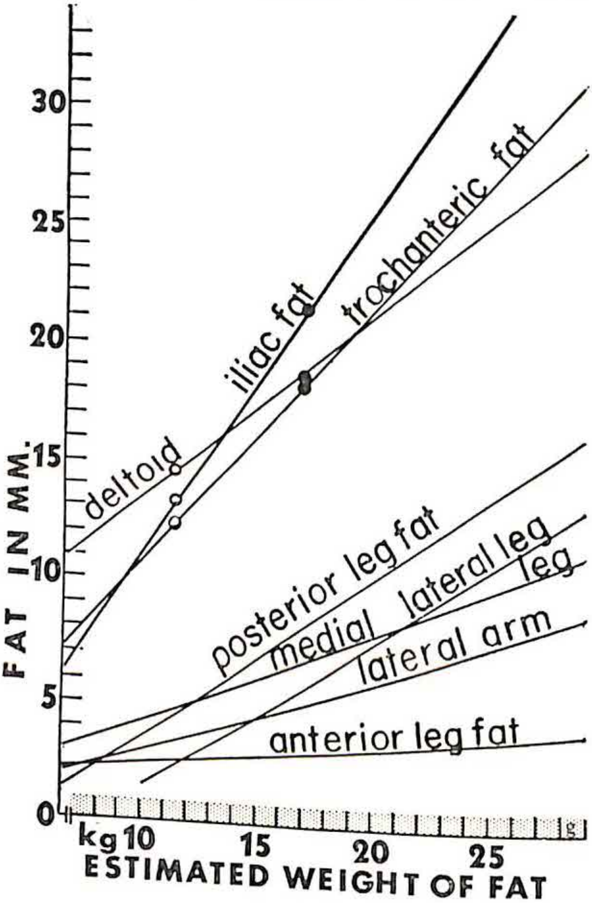

FIG. 1.

Relationships between the estimated weight of fat (abcissa) and the thickness of the subcutaneous fat (ordinate) as derived from data on adult males weighing 58 to 94 kg. Note the tendency for the regression lines to form two clusters, and the crossing over of deltoid and iliac subcutaneous fat. The open circles, representing means for the 20-30 age groups, and the closed circles representing the 50 to 60 age groups, show that the age-accumulation of subcutaneous fat follows the general fat-thickness, fat-weight trend. Therefore it is assumed that the age changes closely resemble simple fattening.

increase in the thickness of fat-containing tissue over the entire body, with the largest absolute gains over the pelvic girdle. Quantitatively the increases over a three-decade period range from negligible values to nearly 8 mm, depending on the part considered. Percentagewise, subcutaneous fat gains range up to 58%, with an average increase of 30% for the 9 fat measurements. In a rough way, there is a division between centrally-deposited fat (which adds at a rate approximating 1 mm per kilogram of weight or fat gained) and peripheral fat, which accumulates at a much slower pace (*cf.* Škerlj *et al.*, '53). The simplicity of this dichotomy is marred by the deltoid-insertion fat, however, and by the markedly different rates of fat accumulation on the 4 quadrants of the lower leg.

In these active white males, the bulk of the increase in weight and subcutaneous fat takes place early, being largely completed by the end of the fourth decade. This parallels the disproportionate early weight gain observed in male coronary heart disease patients and controls (Garn, Gertler, Levine and White, '51), a trend also observable in the U. S. Army 40-105 age-weight norms (Levy *et al.*, '46, p. 952) in Davenport's 1923 summary, and in the Irish data of Hooton and Dupertuis ('52, p. 9). Brožek ('52, p. 787) comments on a similar trend in percentage fat during the same age span, and suggests a parabolic equation as the best fit. However, earlier data, such as Quetelet's (reprinted by Thompson, '44) seem to show a much earlier termination of the period of weight gain, and therefore the question of the type of equation may be left open.

Why fat is gained after osseous maturity is far from clear, although this pattern holds both for man and his domestic animals as well (Clarke and McMeekan, '52; Barnicoat and Shorland, '52). In discussing the greater fat content of ewe and wether carcasses, in comparison to lamb carcasses, Clark and McMeekan generalize as follows: "It is clear that the changes with weight are in accord with our knowledge of the laws of differential growth in the animal body. Bone, as the earliest developing tissue, shows the least increase, fat as the latest developing tissue, the greatest increase."

While part of the adult fat gain noted here and in similar studies may well have a developmental basis, current opinion relative to man stresses the nutritional aspect. This is not simply a matter of increased caloric intake, for fat storage may be speeded by a decrease in energy expenditure as well. Thus, the appetite in the pre-adult male and in the young adult may be physiologically determined, but as appetite becomes a matter of habit rather than need, and as voluntary activity declines, the excess of caloric intake over caloric expenditure leads to the accumulation of fat. In terms of the present material, voluntary activity may decline during the third and fourth decades, becoming more nearly stable thereafter.

But the changes in body composition observed here and in the publications of the Minnesota group (see Brožek, '53 for bibliography) represent changes during a life phase marked by a decreasing hormonal output. From the third decade on, the excretion of urinary 17 ketosteroids is on the decline (Hamilton, '47; Pincus *et al.*, '54). This is true of the 3 and 20 ketosteroids as well, and may be true of the 11 ketosteroids (Clark and Garn, '54b). In a slow way, this gradual falling-off parallels the abrupt changes following castration, with creatinuria, loss of muscle, decreased basal oxygen consumption, and a usual but not invariable increase in fat (Hamilton, '47). Notably, certain steroids have a direct effect on fat metabolism, rapidly decreasing stored fat in castrate animals (Kochakian, '46). And the urinary ketosteroids in the human male appear to contain a substance directly related to energy metabolism (Clark and Garn, '54a). All of these facts lend credence to the possibility that the age-associated increase in fat in the male is in part related to the decrease in hormonal output. This relationship may be direct, involving some degree of hormonal control of fat deposits, or it may be indirect, operating through a decreased energy expenditure.

While there is no doubt that fat is gained during this age span, as can be demonstrated in a variety of ways, it would be a mistake to consider the acquisition as merely a gain in saponifiable fatty acids. More precisely, what is gained is *fat-containing tissue*. This is especially important in studies measuring the thickness of the fat shadow on skiagrams, such as the present one, and where skin-fold thicknesses are measured, for fat-containing tissue also includes connective tissue, a vascular network and water (*cf.* Barnicoat and Shorland, '52). And, while the specific-gravity technique more nearly approaches the measurement of pure fat, the specific gravity of the tissues lost on a weight-reducing regime exceeds the specific gravity of pure fat (Behnke, '53, personal communication; Keys and Brožek, '53, p. 292).

A further complication is offered by the fact that fat itself is not a chemical constant. Neglecting brown fat, which occurs only during embryonic life in man (Wertheimer and Shapiro, '48) there is a notable change in the chemical composition of the fat as age progresses.

This involves a replacement of "soft" fat, with its higher proportion of unsaturated fatty acids, by "hard" fat, with its greater proportion of saturated fatty acids (Barnicoat and Shorland, '52). Hence, the increase in fat-containing tissue observed in this study may be paralleled by a change in the composition of the fat as well. And further, it must be noted that fat in different parts of the body contains markedly different amounts of vitamin E (Quaife and Dju, '49; Quaife, Swanson, Dju and Harris, '49) so that the total amount of alpha tocopherol stored in the body depends both on the amount of fat and the location of the fat. There is a suggestion here that particular patterns of fat deposition may prove to be physiologically meaningful rather than just the actions of a capricious Nature.

Recognizing then that we are concerned with fat-containing tissue, variable in its fat content, in the chemical composition of the fat it contains, and in the substances dissolved in the fat, it is still possible to deal with the variations in the *tela adiposa* as a prime variable in morphological patterning. The different rates at which fat deposits on separate parts of the body make intelligible the changes in surface conformations that may be observed with advancing years and, equally, the different outlines of men, fat and thin. We may predict, from a knowledge of the fat content, the thickness of the fatty tissue on different parts of the body. We may draw grid transformations illustrating the changed outlines that follow changes in the body fat.

SUMMARY

1. The thickness of the subcutaneous fat-containing layer at 9 sites was measured on soft-tissue teleoroentgenograms of 85 males aged 20 to 69 years
2. Eight of the 9 thicknesses showed age-associated increases with an average increase over 4 decades of 30%.
3. The maximum increase was exhibited by iliac fat, being 7.8 mm (58%).
4. Iliac, trochanteric and deltoid-insertion fat were initially thicker and increased more than fat on the lower arm and leg.
5. The weight of fat, as estimated from trochanteric fat, increased from 11.5 to 17.3 kg and estimated percentage fat rose from 16% to 22%, paralleling the rise in weight.

6. Both fat and weight increases were more marked during the third and fourth decades, as seems to be characteristic of other recent data on white males.
7. As shown on fat-thickness, fat-weight curves, changes in surface contours resemble simple fattening.
8. Attention was directed to the fact that these increases in fat-containing tissue involve significant additions to non-fatty components and changes in the chemical composition of the fat.
9. The fact that fat increases while the level of excreted ketosteroids decreases suggests either possible hormonal mediation or indirect mediation through decreased voluntary activity.
10. The simple relationships existing between the estimated weight of fat and the 9 fat thicknesses suggest an approach to one aspect of body form.

ACKNOWLEDGEMENTS

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BOOK REVIEWS

Modern Learning Theory. By W. K. ESTES, S. KOCH, K. MACCORQUODALE, P. E. MEEHL, C. G. MUELLER, JR., W. N. SCHOENFELD, W. S. VERPLANCK. Ed. by A. T. POFFENBERGER. xv + 379 pp. Appleton Century-Crofts, Inc., N. Y. 1954. $5.00.

This is a review of the handiwork of seven reviewers who examined the work of 5 psychologists considered to occupy a prominent position in the field of learning. Hull gets 177 pages, Tolman 90, Skinner 56, Lewin 28, Guthrie 32.

We are led over hill and dale of the Hullian system by Koch who concedes that "we have done what may be construed as a very nasty thing." He concludes that Hull "failed not merely because he aimed at comprehensive theory, or because relevant empirical knowledge is too painfully slim to justify even far more limited attempts" but "because he did not adequately meet concrete problems of *empirical definition, of measurement, of quantification, of intervening variable function construction,* and various sub-specifications of all of these." We wonder, though, why it took Professor Koch nearly one-half of the book to come to this conclusion.

Next Tolman is taken for a ride by Professors MacCorquodale and Meehl, ending with an "Apology to Tolmanites and Others" and the statement: "In its main outlines, we have seen no reason to criticize Tolman's approach as a *knid* of behavioral theory. Like its other critics, we wonder at its lack of even a minimal amount of formalization over the 20 years of controversy and experiment."

It is a relief in the next two chapters dealing with Skinner and Lewin to be through with "apologies" and to hear some straightforward simple talk. Together these two authors take up on 20% of the book, but contribute most of the sense. Verplanck says: He [Skinner] wishes to find out how animals behave and seeks a vocabulary that will let him talk about how they behave. Because of the existence in Sherrington and Pavlov of sets of data of the kind he believes are needed, he has adopted many of their terms and applied some of their laws in defining his area." A good comparison is made of Skinner, Tolman and Hull. Skinner makes good sense and is head and shoulders above the others.

Estes in 27 pages gives us an equally lucid analysis of Lewin's concepts. The fallacies of the popular "field theory" are revealed. "None of the so-called field theories have been developed in conjunction with programs of experimental research upon learning. Typically the field theorists have developed their systems in other areas, then have attempted to collect the psychology of learning as an extra dividend without much additional investment."

Under the pens of Mueller and Schoenfeld, Guthrie emerges as a pygmy strutting among the concepts of the more illustrious folk in psychology (Pavlov, Thorndike, Lashley).

Stimulated by the abstruse formulation and the elaborate diagramatics of the first two chapters, I made a simple statistical survey of the references in the bibliographies of the 5 reviewers. To the great figures in the history of learning and psychology, or those that I consider prominent, *viz.*, James, Pavlov, Bechterev, Thorndike, Watson, and Koehler, there are no references with the exception of one to Pavlov by Verplanck and one to Koehler by Estes. This seems to me some criterion of the scope of the reviews.

The book will undoubtedly appeal to the academic psychologist who wants to have a thorough digest of these 5 contemporary authors of learning theory, but it will not satisfy the general reader who is looking for a broad and scholarly discussion.

W. HORSLEY GANTT

BOOKS RECEIVED

Needed Research in Health and Medical Care: A Bio-Social Approach. By Cecil G. Sheps and Eugene H. Taylor. ix + 216 pp. University of North Carolina Press, Chapel Hill, N. C. 1954. $5.00.

Free and Unequal: The Biological Basis of Individual Liberty. By Roger J. Williams. xiii + 177 pp. University of Texas Press, Austin, Texas. 1953. $3.50.

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Cultural Difference and Medical Care. By Lyle Saunders. 317 pp. Russell Sage Foundation, New York, N. Y. 1954. $4.50.

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SKIN-FOLD CHANGES WITH INCREASING OBESITY IN YOUNG AMERICAN MALES *

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INTRODUCTION

DURING a recent anthropometric examination of approximately 2,000 young American men, soon after induction into the United States Army and before basic training, skin-fold measurements were obtained. The availability of skin-fold data on such a large series and yet one which was remarkably homogeneous in terms of age and physical selectivity makes it possible to conduct statistical comparisons of subsamples within this group with reasonable validity. The basic description of the skin-fold and related measurements and their demographic distribution will be treated in another report. Two problems are examined here: (1) the question of utilizing skin-fold measurements obtained on American Negroes to calculate their body fat; and (2) whether data on large numbers of men of varying degrees of obesity shed any further light on the relationship between subcutaneous fat and total obesity.

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* Presented at the annual meeting of the American Association of Physical Anthropologists, Yellow Springs, Ohio, March 28, 1954.

It is not the purpose of this paper to review the history and theory of skin-fold measurements as indicators of obesity; this has been covered in a series of recent review articles (Brožek and Keys, '51; Brožek, '53; Keys and Brožek, '53). However, a brief statement of some of the knowns and unknowns is pertinent to the problems presented here. The most interesting aspect of subcutaneous fat as measured by skin-fold thickness is in its relationship with a calculation of total body fat obtained by densiometry. Essentially, this technique consists of a close statistical association between the skin-fold thickness at selected sites and body fat (as a percent of weight) calculated from the average density (weight divided by body volume). Brožek and Keys ('51) have demonstrated such a close relationship for 133 University of Minnesota college men that a multiple regression equation based on the skin-fold thickness at the chest, arm, and abdomen can be used to calculate body fat with a high degree of reliability. There are a number of limitations inherent in the use of this equation, only two of which will be discussed here: (1) since this equation was based on White college students, can it be applied equally to other racial groups, in this case American Negroes? (2) since it is a single multiple regression equation which fits all young men whether lean or obese, it assumes that the skin-fold thicknesses at all sites represented in the equation increase linearly with increasing obesity. Is this true when a large sample is analyzed? Unfortunately, one vital portion of the data on the Army men, a reliable and independent measure of body volume from which average density could be calculated, was not available. None of the densiometric techniques now available or expected in the near future (*e. g.*, underwater weighing, air displacement, gas dilution), could handle such large numbers of men in the time available. This lack of an independent method of calculating body fat limits the analyses to the observed data, skin-fold thickness, body fat calculated by the Brožek and Keys' equation, and total body weight.

Some of the pertinent elements of the measuring technique are reported here because the increasing number of measuring instruments and skin-fold sites in use might otherwise eventually result in the grouping of basically inapplicable data. Five skin-fold sites were measured on these subjects: chest—above and to the right of the right nipple; arm—on the back side, half-way up the upper arm; back—immediately below the right scapula; abdomen—to the left of the navel; and knee—above the right kneecap. The subjects were measured in an upright position by two technicians and their average value was used. Each investigator measured every skin-fold site at least three times before proceeding to the next area. The calipers used were a slightly modified version of the Minnesota type skin-caliper similar to that described by Brožek ('52) and illustrated by Newman ('52). The caliper points had a diameter of 6 mm with a surface pressure of approximately 5 g per sq mm.

The series used here were 1,702 White males and 292 American Negro males. This latter group will be referred to subsequently as Negroid to acknowledge the obvious racial admixture which was present in this sample. The average age of these men was 20.7 years with a relatively restricted age range, 17 to 28 years. The effect of physical selection on these men is unknown, but it probably deviates no more from a truly random sample of young American men than a series based on volunteer college students.

NEGROID-WHITE COMPARISONS

Any detailed comparision of White and Negroid skin-folds is complicated by the fact that the Negroids are superficially far leaner than the Whites. For example, the average Negroid body fat calculated from Brožek and Keys' regression equation was 3.9%, approximately half the comparable white value of 6.8%. This preponderance of small skin-fold values in the Negroids results in a lack of individuals whose skin-fold values can be compared with the obese White subjects. This paper is not concerned with why the Negroids show lower skin-fold values but whether their subcutaneous fat is relatively rather than absolutely comparable to Whites in terms of gross measured quantities.

One method of investigating the relationships between skin-fold sites, irrespective of the amount present, is to calculate correlation coefficients for the 5 areas. The 10 possible coefficients are shown in table 1 for both racial groups. It is apparent that Whites and Negroids show a similar arrangement of coefficients in bodily areas; the chest, back, and abdomen skin-folds are highly intercorrelated with each other and less correlated with the extremities (arm and knee) which form another correlated set. The two matrices of coefficients are very close in magnitude, but the Negroid coefficients are slightly lower in value. There does not seem to be any readily apparent reason for this difference unless it is the effect of racial mixture appearing as heterogeneity in the coefficients.

A statistical test of significant difference between the comparable White and Negroid correlation coefficients was applied to the 10 pairs given in table 1. Since the numbers of men involved for each site relationship were constant, a common significance level of *t* of 1.96 at the 5% level and 2.58 at the 1% level were used. The results are shown below in table 2. It is difficult to evaluate such a table where two relationships show significant difference and 8 do not, but it would appear that the skin-fold relationships between the bodily areas are at least roughly similar in these groups. A possible explanation for the difference in chest-abdomen coefficients may be brought out in the next section.

TABLE 1

Correlation Coefficients between Skin-fold Sites of Measurement

	ARM	BACK	KNEE	ABDOMEN
WHITES				
Chest	.79	.90	.73	.90
Arm		.78	.83	.81
Back			.74	.87
Knee				.75
NEGROIDS				
Chest	.79	.80	.67	.86
Arm		.74	.82	.80
Back			.68	.86
Knee				.72

Lacking any measure of body volume, it was not possible to construct regression equations between skin-fold values and calculated body fat for these two groups and to compare them. Furthermore, it was not possible to throw much light on the question of racial differences in body fat from a regression equation based on Whites alone. Because of this postulate, the samples were divided into increments of apparent obesity on the best evidence available, a summation of the measured skin-folds. The term "apparent obesity" is used for two reasons: (1) It must be assumed that the two principal fat depots, superficial (subcutaneous) and deep (largely visceral), increase in roughly equal proportion with increasing obesity. This has been shown in Whites by the close correspondence between skin-folds and tissue density, but it remains an assumption for the Negroids. (2) It is apparent from Brožek and Keys' ('51) prediction equation between skin-fold thickness and body-fat from densiometry that the various sites do not have equal weight in this association, for example, abdomen, chest, and arm have relative values of 15, 39, and 46%, respectively. No differential weightings could be used with this Army data without prejudicing the results, and it is believed that no large error has been introduced by the simple addition of the skin-fold measurements into a summation value which must roughly align the men into increments of increasing obesity.

TABLE 2

Comparison of White and Negroid Values of r

SITE	"t" VALUE	SIGNIFICANCE
Chest-arm	0.16	None
Chest-back	5.47	1%
Chest-knee	1.88	None
Chest-abdomen	2.81	1%
Arm-back	1.41	None
Arm-knee	0.62	None
Arm-abdomen	0.47	None
Back-knee	1.72	None
Back-abdomen	1.25	None
Knee-abdomen	0.94	None

The White and Negroid samples were divided into 5 mm intervals based on the sum of abdominal, arm, and chest skin-folds. The mean value for each of the three skin-fold sites for all men in each summation interval was calculated and plotted in figure 1. The entire sample of 1,702 Whites and 292 Negroids is not represented in this figure which shows only those individuals whose combined skin-fold value totaled 45 mm or less. Above this value there were not enough Negroids to avoid erratic sampling errors. The actual numbers of men represented by the plotted values in figure 1 are given in appendix A.

The lines of mean skin-fold values in this figure closely approximate regression lines and may be tested statistically for significant differences in slope. This was accomplished for each racial pair and the lines were found to be not significantly different. It is apparent, however, that the Negroid has low skin-fold values in chest and arm and accompanying high abdominal skin-fold thickness as contrasted with the Whites. These differences were visible at the time of measurement. The Negroids were characterized by well-developed and well-defined musculature in the torso and upper extremities, and musculature could not be well-defined when covered by a thick layer of subcutaneous fat. Hooton ('48) mentioned this when comparing body build photographs in 3,000 Negroids and 30,000 Whites. He said that "the shoulders of the Negro customarily show relatively greater muscular development than is found in Whites.

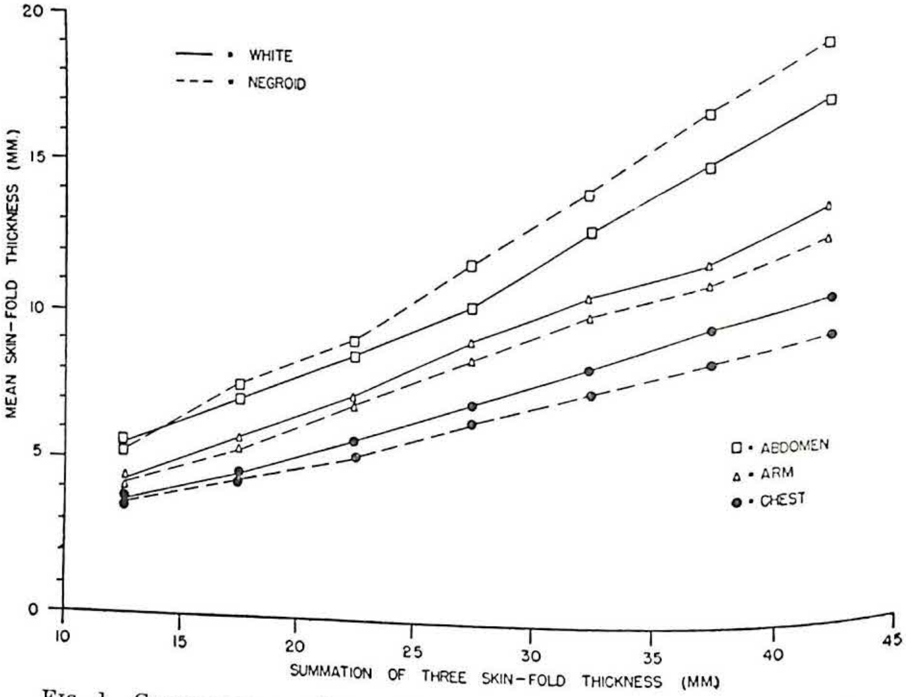

FIG. 1. COMPARISON OF THREE WHITE AND THREE NEGROID SKIN-FOLDS.

The Trapezii and Deltoids are characteristically well developed, as are also the pectoral muscles, the abdominal muscles, and those of the back. Thigh muscles are also generally good."

The minimal values for men who have 10 to 15 mm total for the three skin-fold sites are remarkably consistent between the racial groups. Two points may be inferred from this: (1) the average value must be very close to fat-free, doubled skin-fold thickness; (2) it suggests that there is no measurable difference in fat-free skin-fold thickness between Negroids and Whites, at least at these particular sites. Tanner ('51) records three millimeters as the minimum skin-fold thickness found in approximately 400 British students. No skin-fold reading of a lower magnitude was recorded for the 2,000 men reported here.

It is possible to estimate the amount of body fat by substituting the mean values given in figure 1 for any given interval in the Brožek and Keys' ('51) prediction equation. This assumes that the overall subcutaneous fat to average body density relationship is basically similar for the two groups, and further that the Negroid superficial versus deep fat ratio is the same as that found in the White subjects. A calculation of percent body fat using the mean skin-fold values, rounded to whole millimeters, for the largest interval used in figure 1 (40 to 45 mm) gave a White value of 8.4% and a Negroid value of 7.9%. One half of one per cent is a very small difference for a measurement as full of potential pitfalls as the estimation of body fat.

The first question asked in this study, whether skin-fold measurements on American Negroes can be converted to estimates of per cent body-fat from relationships established on White subjects, cannot be answered definitively from the data presented here, but it appears that no great error would result from such a practice. The relationships shown in table 1 and figure 1 are considered sufficiently similar to warrant using the total series of 1,994 men in examination of the next question, whether data on a large group of young men can show any new relationships between skin-folds and obesity.

MEAN VALUES FOR THE COMBINED SERIES

The use of the combined series, which now might be categorized as young American male, offers a much larger range of obesity than could be shown in figure 1 because of the inclusion of the fatter White subjects. The average values at all 5 measured skin-points have been plotted for all the summation of total skin-fold thickness intervals in which 10 or more individuals were represented in figure 2. The largest summation interval used in this figure (130 to 139 mm) represents approximately 20% body fat. The actual number of individuals used in the calculation of the mean skin-fold values for each summation interval in figure 2 are given in appendix A.

There are several relationships to be seen in this figure. The most striking is the curvilinear relationship of the chest skin-fold to increasing total thickness. If this trend continues into the range of even greater obesity, it becomes second only to the abdominal site in importance. Both linear and second degree curve regression equations were calculated on the chest mean values and the standard error of the curvilinear equation was significantly smaller than that of the linear equation. This curvilinearity may be caused by some peculiarity of this series, and, in any case, is not of much practical importance.

Even more interesting is the difference in the slope of the lines across the range of obesity represented here. If a line perpendicular to the abscissa were drawn about midway on the summation scale, in figure 2, it could be considered to divide the series into those leaner than average on the left and those fatter than average on the right. The fatter group on the right half of the figure shows considerably different slopes than characterize the leaner group. The marked inclination of the abdominal line is readily apparent; this trend is present on the left half but is not as distinctive in terms of millimeters of skin-fold thickness. It is also apparent that the chest slope is roughly parallel to that of abdomen and both are at odds with the slope of arm and knee, while the slope of back skin-fold appears to have an intermediate position. This probably should be interpreted as illustrative of a differential development of subcutaneous fat with increasing obesity. The torso, especially on the

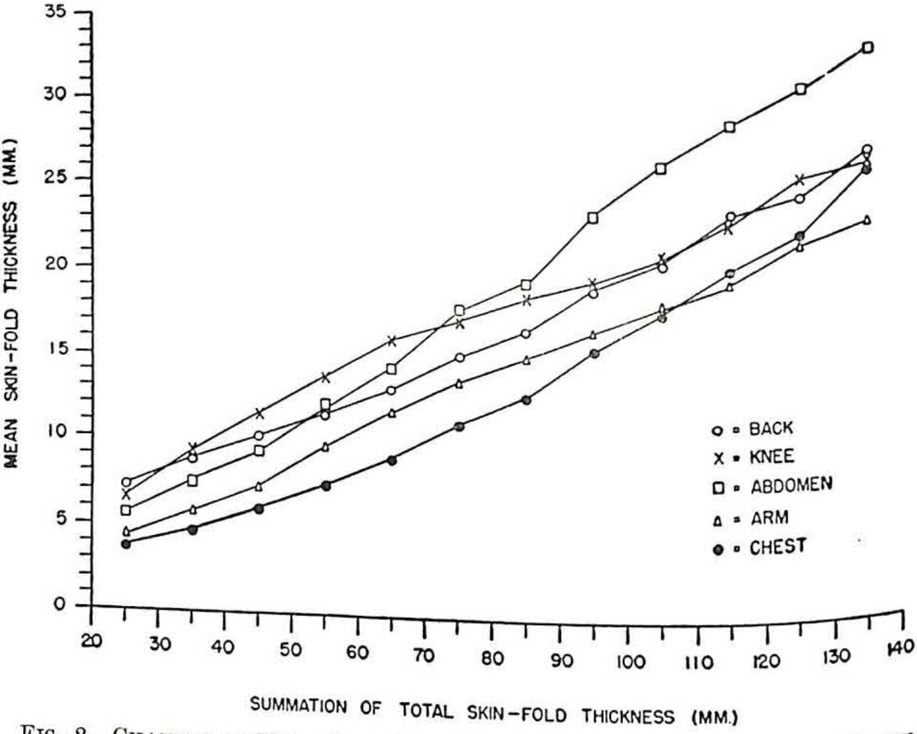

Fig. 2. Changes in Mean Skin-Fold Values with Increasing Total Thickness for Total Series (2000 Males).

anterior aspect, has a larger capacity to store subcutaneous tissue and becomes increasingly important in fatter individuals, while the extremities are limited in the physical space available for this tissue and, therefore, grow less important. If this situation also occurs when a large sample of men are arranged in ascending order of body density, the use of any one multiple regression equation for calculating percent body fat cannot be entirely satisfactory. It probably makes little difference in men of average fat values, but it must decrease the accuracy of prediction on the very lean and the very fat.

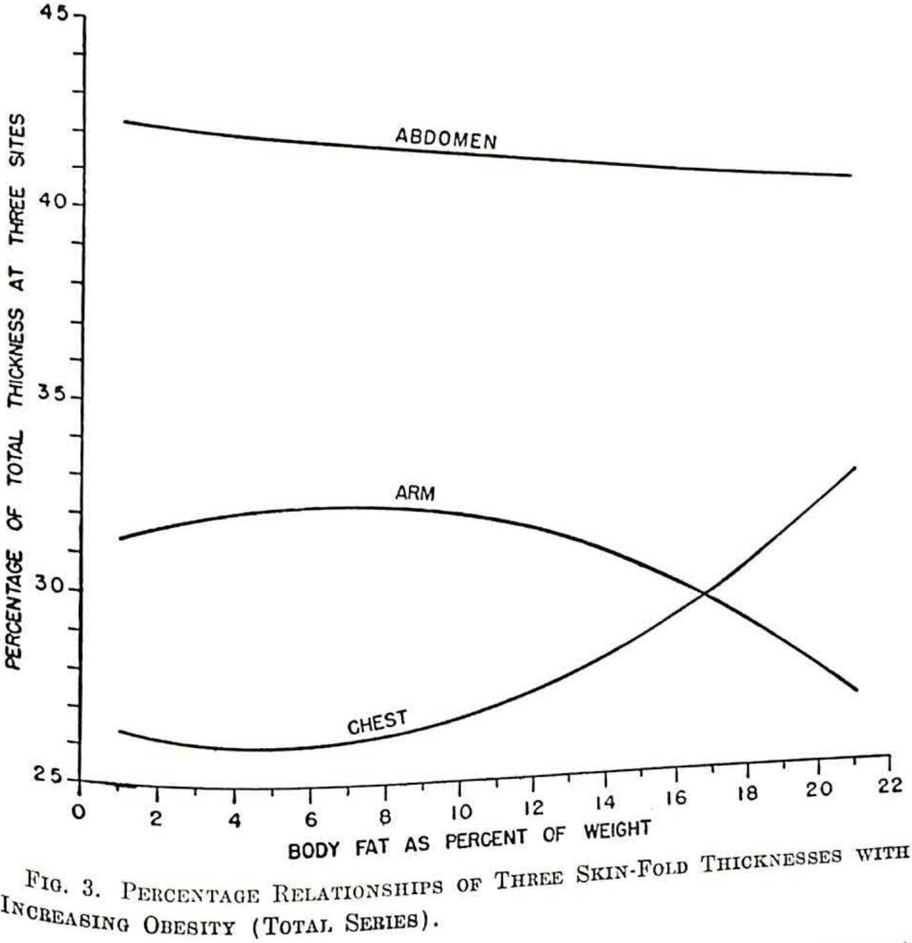

FIG. 3. PERCENTAGE RELATIONSHIPS OF THREE SKIN-FOLD THICKNESSES WITH INCREASING OBESITY (TOTAL SERIES).

One additional graphic method of representing the relationships of the various skin-fold sites with increasing obesity has been used in figure 3. In this graph the mean skin-fold values at abdomen, arm, and chest have been expressed as percentages of the total thickness on the ordinate axis. These percentages have been plotted against calculated percent body fat. Since the body fat data were derived from a multiple regression equation of these three skin-folds, the values of fat and skin-folds cannot be independent of each other.

A plot of percentage values such as shown in figure 3 maximizes any change in proportions at the three sites because as one site value rises some other must decline to preserve a totality of 100% for each interval. Only the calculated lines of best fit have been used in this figure to achieve a simplified graphic effect. The data from which the lines were calculated are given in appendix A. The lines of best fit for arm and chest are obviously curvilinear in this figure. Linear and second degree curve regression equations were calculated from the mean percentage values for each interval and the standard error of the arm and chest second degree equations were significantly smaller than the standard errors of the linear equations.

The arm and chest lines in this figure form a reciprocal pair, similar to the crossover shown in figure 2. This presumably should be interpreted in the same manner, namely, that with increasing obesity subcutaneous fat is deposited in relatively greater quantities in the torso than in the extremities.

SUMMARY AND CONCLUSIONS

Two problems involving skin-fold measurements on a large series of young American men have been examined in this paper. The results cannot be considered definitive because no independent measure of average body density was available; the conclusions arrived at using the sum of the skin-folds can only be termed indicative.

An analysis of Negro and White skin-fold relationships by comparing correlation coefficients between skin-fold sites and average skin-fold values for increments of obesity showed small and not statistically significant differences between these racial groups. It was concluded that the present data do not show any reason why body fat on Negroes cannot be calculated from the published prediction equation based on White subjects. However, the comparison was limited by the scarcity of obese Negroes, and it is possible that the observed Negro deficiency of fat over the pectoral and triceps regions might become practically significant if very obese Negroes were compared to their White counterparts. This racial difference in the regional distribution of subcutaneous fat is an intriguing problem which should repay further investigation.

A graphic comparison of the various skin-fold sites in the combined series of 2,000 men for presumed increments of increasing obesity indicated a change in the proportional representation of the average site values between the lean and fat subjects. The anterior torso areas, chest and abdomen, showed a rise in relative importance with increasing obesity at the expense of the extremity areas, *i. e.*, the arm and knee. This was interpreted to be at least partially the result of physical space limitations in the extremities. It may form a source of error in predicting fat on all men of a comparable age group with a single prediction equation.

This paper has only touched on a few of the interesting possibilities inherent in the skin-fold measurements of these 2,000 young men. Its major purpose was to validate, if possible, the calculation of body fat on the 361 Negroids for use in other analyses. It is believed that this has been accomplished, and a new and intriguing field of anthropometric racial investigation can be developed through the skin-fold measurement technique.

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APPENDIX A

Additional Data for Figures 1, 2, and 3

FIGURE 1			FIGURE 2			FIGURE 3				
mm	Frequency		mm	Frequency		Per cent		Mean Percentage of Total Thickness		
Skin-Fold	White	Negroid	Skin-Fold	White	Negroid	Body Fat	Number	Chest	Arm	Abdomen
10-14	23	12	25-29	16	8	0-1	74	26.7	30.3	43.0
15-19	201	83	30-34	75	24	2-3	554	25.8	32.0	42.2
20-24	345	88	35-39	175	56	4-5	490	26.1	33.1	40.8
25-29	266	45	40-44	203	56	6-7	321	26.1	32.9	41.0
30-34	230	21	45-49	181	39	8-9	194	25.6	33.0	41.4
35-39	157	18	50-54	172	25	10-11	118	27.4	31.4	41.2
40-44	111	10	55-59	151	19	12-13	76	27.7	30.7	41.6
			60-64	150	19	14-15	58	29.5	28.8	41.7
			65-69	92	13	16-17	36	29.1	29.4	41.5
			70-74	75	10	18-19	26	31.4	28.2	40.4
						20-21	22	32.5	27.6	39.9

AGE DIFFERENCES IN THORACIC FORM AS INDICATED BY THORACIC INDEX

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MARTIN ('28) and Mollison ('33) explain the flatter build of the human thorax, compared with that of other primates and mammals, by the change to upright position. This phylogenetic tendency to a flatter thorax is also seen in ontogenesis. The human thorax becomes flatter during the fetal and postnatal growth periods (Martin, '28). We have followed the transformation of the form of the thorax in Japanese in all the age periods of postnatal life.

The present study is based on body measurements of 9369 inhabitants of the Tōhoku area, especially Tsugara district of Aomori Prefecture in Japan. These individuals, from birth to 85 years of age, were measured for stature, weight, chest depth, breadth and girth, and some other measurements. Chest dimensions were taken at the end of expiration of tidal air. The percentage of chest depth to chest breadth is taken as the thoracic index, although Martin used the inverse.

The subjects were mostly rural inhabitants. Children from 6 to 11 years old were primary school pupils and those from 12 to 14 attended middle school; these are periods of compulsory education in Japan. The subjects from 15 to 17 were high school pupils and almost all those from 18 to 20 were also students. The adult group consisted of farmers, teachers, officials, and service workers, and the people over 60 years of age had had various careers and were mostly inmates of an institution for the aged.

FINDINGS

The findings are shown in table 1 and in figure 1. The average thoracic index of the newborn is 84 in both sexes. After birth the index decreases rapidly until in 4-year old children it is about 72 in boys and

TABLE 1

Stature and Thoracic Index by Sex and Age
(means and standard errors)

	MALE			FEMALE		
Age	Number	Standing Height (cm)	Thoracic Index	Number	Standing Height (cm)	Thoracic Index
0–6 mos	74	63.4±0.49	83.88±1.37	71	62.1±0.47	83.72±0.94
6–12	88	69.0±0.35	80.28±0.76	86	68.2±0.44	79.80±0.68
1–2 yrs	142	76.3±0.38	78.24±0.44	131	74.3±0.33	77.96±0.60
2–3	82	85.2±0.51	76.72±0.57	79	82.1±0.53	75.40±0.50
3–4	83	91.2±0.51	74.02±0.45	77	90.7±0.43	72.61±0.66
4	33	100.2±0.92	72.24±0.79	31	98.5±0.98	71.00±0.84
5	36	104.5±0.73	71.88±0.66	36	103.5±0.76	70.70±0.82
6	115	109.1±0.36	72.14±0.37	132	108.8±0.36	70.68±0.39
7	175	114.1±0.32	72.16±0.33	166	113.6±0.37	70.38±0.41
8	181	119.9±0.37	71.46±0.16	185	118.3±0.35	70.05±0.32
9	181	125.1±0.37	71.52±0.33	174	123.9±0.37	70.34±0.36
10	212	129.4±0.34	71.12±0.30	180	128.6±0.43	69.76±0.35
11	185	133.4±0.36	71.76±0.34	189	133.8±0.47	69.18±0.32
12	168	138.5±0.49	71.90±0.33	159	141.6±0.55	68.98±0.37
13	321	144.6±0.42	71.64±0.25	272	145.1±0.39	71.06±0.28
14	258	151.4±0.46	72.16±0.26	286	148.8±0.32	71.48±0.35
15	506	158.2±0.31	70.13±0.24	380	151.2±0.25	71.21±0.27
16	434	162.0±0.26	69.20±0.25	275	152.9±0.27	71.28±0.34
17	377	163.2±0.28	68.98±0.23	198	153.5±0.33	71.62±0.40
18	322	164.4±0.28	70.08±0.30	184	154.3±0.39	72.24±0.36
19	289	165.5±0.31	68.92±0.30	89	154.0±0.51	72.02±0.56
20	191	165.1±0.38	68.80±0.43	43	153.2±0.64	71.16±0.94
15–19*	91	161.3±0.68	69.20±0.50	66	151.3±0.61	70.64±0.48
20–24	170	164.9±0.39	68.52±0.44	123	152.1±0.48	72.00±0.60
25–29	127	164.7±0.53	67.92±0.59	87	151.6±0.64	72.76±0.60
30–39	179	162.4±0.48	69.48±0.39	102	149.2±0.57	72.98±0.50
40–49	158	162.3±0.54	70.24±0.47	91	148.8±0.64	74.62±0.61
50–59	141	159.6±0.71	72.18±0.53	67	145.9±0.91	77.02±0.71
60–69	18	(154.0±1.60)	76.67±2.06	17	(144.4±1.57)	82.05±2.43
70 & over	26	(152.5±1.67)	80.38±1.68	30	(140.2±1.61)	83.33±1.78

N. B. Subjects from 6 to 11 years are primary school children, from 12 to 14 pupils of middle school, from 15 to 17 pupils of high school and from 18 to 20 also mostly students.

* 15–19 year old youths who do not attend school.

71 in girls. It seems that the decrease is greater in girls. The average thoracic index continues to decline in girls 4 to 12 years of age when it is about 69, significantly lower than in boys of the same age. In other studies of Japanese (figure 2) and in American Negroes (figure 3) the thorax is also flatter in females of these ages.

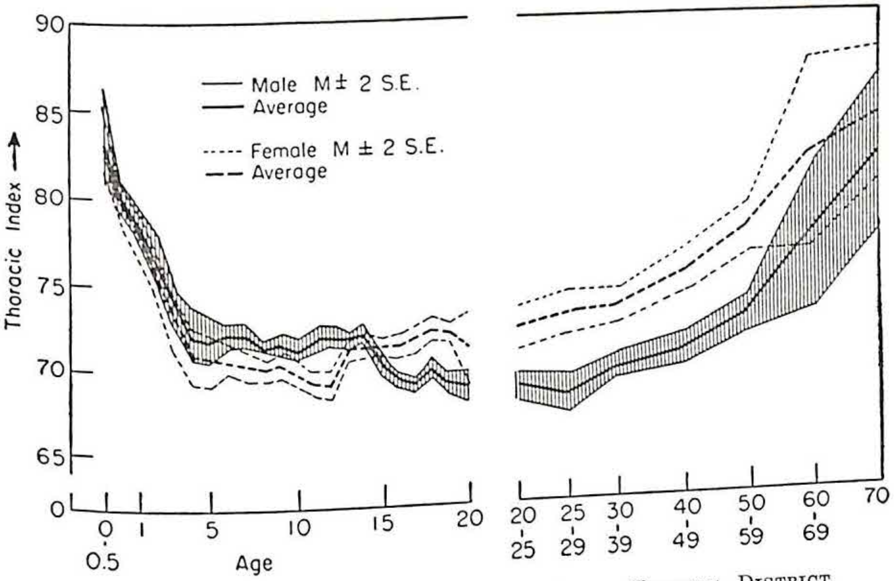

FIG. 1. THORACIC INDEX CURVE OF JAPANESE IN TSUGARA DISTRICT.

The thoracic index of girls age 13 appears to be suddenly larger (to about 71) while the index of the males goes down from 15 to 25–29 years of age (to about 68) so that the male chest is flatter from between 14 and 15 years of age onward. From about the age of 25 to 29 chest index begins to be larger again, increasingly so after the 40–49 year old period and this is apparent in both sexes. In the age class of 70 and over (really 70–85 years of age) the index is again over 80 in both sexes as in the newborn.

DISCUSSION

The chest depth and breadth of Japanese have also been measured by some other authors (figure 2), but not for all age classes. Differences in the thoracic index reported by different authors seems to be partly explained by differences in the province, occupation or rural-urban nature of the population studied; it may also represent differences in technique involving different phases of respiration or a different level of the chest in the measurements of different authors. In any case, all authors report the same tendencies as to sex and age differences. The other studies of Japanese accord with ours in age groups up to 11 or 12

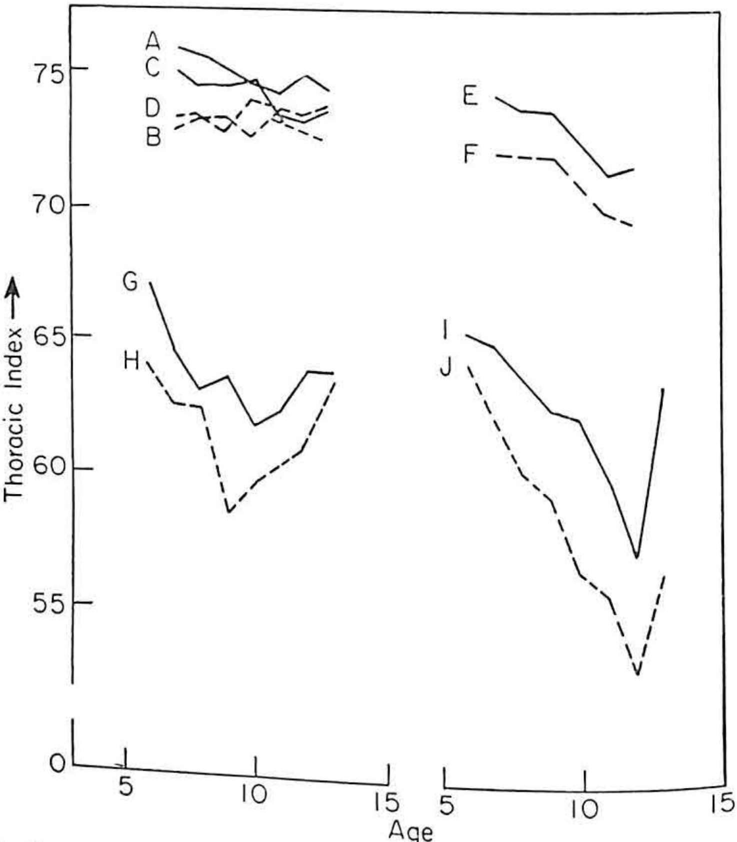

FIG. 2. THORACIC INDEX CURVE OF JAPANESE SCHOOL CHILDREN BY OTHER AUTHORS.

Solid line: male; dotted line: female

(a) (b) (Urban) MINAYOSHI, T. 1928, Nippon Gakkō Eisei (in Japanese), *10*: 721-742.
(c) (d) (Rural) MINAYOSHI, T. *Ibid.*
(e) (f) SHIMA, S. 1931, Nippon Gakkō Eisei (in Japanese), *19*: 199-204.
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(i) (j) INAGAKI, T. AND A. NAKAZAWA 1947, *Ibid.*, *11*: 55-165.

years of age, but Martin's ('28) data on Negroes is most similar to ours in all age classes. All groups seem to show that the rounder chest of the newborn becomes increasingly flatter until about 4 years of age, that before puberty girls have relatively flatter chests than boys and that after puberty girls' chests become markedly rounder. After 14 years of age the period of compulsory education ends and few children of the less privileged classes attend school; there is, therefore, a selective factor in our series. Nevertheless, there seems to be a real tendency to further flattening in the chest of males until 25–29 years. The increased flattening of the chest in successively older persons up to this age seems to be a developmental phenomenon. Although chest depth also increases, the increase in chest breadth is more rapid. Martin ('28) says that chest breadth always increases at the cost of chest depth. Chest breadth shows a significant positive correlation with stature, but chest depth does not (table 2). The greater relative depth of the female chest after puberty seems to represent a real sex difference; it is, of course, at this time that there is a rapid development of the female breasts.

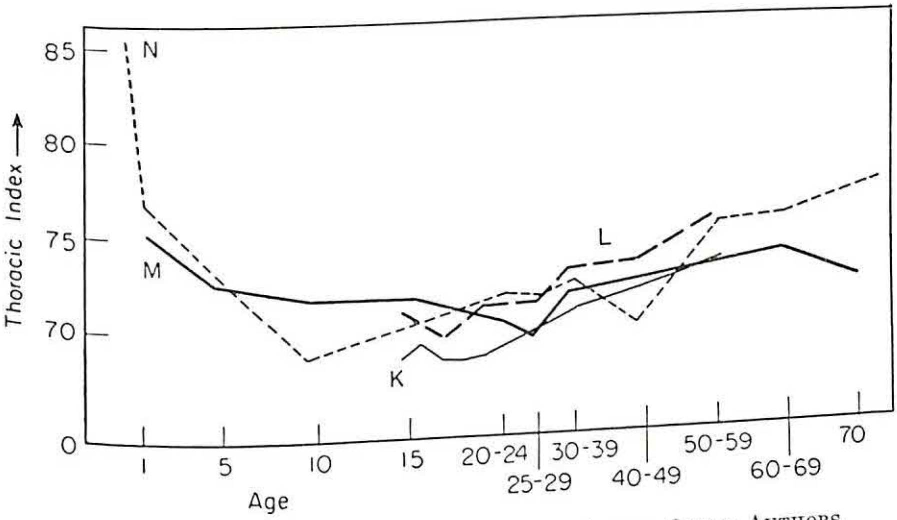

Fig. 3. Thoracic Index Curve by Sex and Age by Other Authors.
Solid line: male; dotted line: female.
(k) (l) Japanese prisoners, Dohi, N. 1949, Essays of Anthropology, Human Genetics and Constitution (in Japanese), Keiō Gizyuku Univ., *14*: 15-206.
(m) (n) American Negroes, Martin, R. 1928, Lehrbuch der Anthropologie, Gustav Fischer, Jena.

Many authors agree with us in noting a cessation of growth in chest breadth after 30–39 years of age but a continued slow increase in chest depth. The thorax thus becomes rounder, but this may be a regressive rather than a developmental phenomenon.

In order to analyze the thoracic index further, we calculated separately that of adults of different occupation groups (table 3). In both males and females the lowest index is found in teachers and progressively higher indices are found in officials, service workers and farmers respectively. This same tendency is seen in the different age periods considered separately, and, conversely, the tendency to an increase in the thoracic index with age occurs in each occupational group.

TABLE 2

Coefficients of Correlation between Diameters of Chest, Thoracic Index, Relative Transverse Area of Chest, and Other Physical Measurements of Adults

	CHEST DEPTH		CHEST BREADTH		THORACIC INDEX		RELATIVE TRANSVERSE AREA OF CHEST	
	Male	Female	Male	Female	Male	Female	Male	Female
Standing height	0.040	0.017	0.245**	0.130*	—0.184**	—0.121*	0.106**	0.165**
Sitting height	0.022	0.025	0.250**	0.158**	—0.193**	—0.122*	0.104**	0.137**
Girth of chest	0.583**	0.622**	0.628**	0.600**	0.087*	0.110*	0.695**	0.679**
Body weight	0.544**	0.563**	0.616**	0.574**	0.009	0.198**	0.628**	0.646**
Pelidisi	0.494**	0.504**	0.390**	0.421**	0.193**	0.190**	0.567**	0.809**
Vital capacity	0.130**	0.167**	0.372**	0.327**	—0.037	—0.026	0.311**	0.276**
Strength of back muscles	0.110*	0.149*	0.230**	0.247**	—0.036	—0.003	0.259**	0.419**

N. B. Number of measured adults: 656 males and 350 females.

* Over the level of significance of $P = 0.05$.

** Over the level of significance of $P = 0.01$.

The relative thickening of the chest in older age may be caused by the increased spinal curvature or by the decrease in height of the thorax which follows the loss of fluid and compression of the *disci intervertebrales.*

The thorax is composed of the spine (vertebrae), ribs and sternum. The spine is the most stable of these three elements and the motility of the ribs increases from the vertebral to the sternal end. Chest breadth is less affected by movement than is chest depth. The difference between measurements on inspiration and on expiration is less for chest breadth than for chest depth (table 4). Furthermore, chest depth tends

TABLE 3

Chest Depth, Breadth and Thoracic Index by Sex and Occupational Group
(means and standard errors)

SEX	AGE CLASS	TEACHERS	OFFICIALS	SERVICE WORKERS	FARMERS
Male					
Chest depth (cm)	20–29	18.03±0.11	18.15±0.15	18.44±0.23	18.98±0.21
	30–49	18.64±0.12	18.63±0.16	19.10±0.30	19.15±0.21
	50–59	18.99±0.25	19.32±0.24	19.30±0.25	19.40±0.28
	Average	18.55±0.06	18.70±0.07	18.95±0.07	19.16±0.08
Chest breadth (cm)	20–29	26.63±0.12	26.45±0.15	26.29±0.29	27.11±0.21
	30–49	26.99±0.13	26.88±0.16	26.77±0.27	27.06±0.20
	50–59	27.04±0.26	26.94±0.24	26.70±0.26	26.40±0.34
	Average	26.89±0.04	26.76±0.05	26.59±0.02	26.86±0.07
Thoracic index	20–29	68.08±0.49	69.84±0.05	69.88±0.91	70.14±1.04
	30–49	69.20±0.52	69.64±0.66	70.92±0.94	71.02±0.97
	50–59	70.06±1.12	72.00±1.11	72.46±1.15	73.54±1.16
	Average	69.11±0.28	70.49±0.34	71.09±0.37	71.64±0.38
Female					
Chest depth (cm)	20–29	16.91±0.14	16.53±0.20	17.80±0.17	17.94±0.25
	30–49	17.00±0.18	17.50±0.18	18.21±0.17	18.52±0.21
	50–59	—	—	(18.47±0.26)	(18.92±0.25)
	Average	16.96±0.08	17.02±0.10	18.02±0.09*	18.23±0.09*
Chest breadth (cm)	20–29	23.72±0.13	23.41±0.21	24.44±0.12	24.56±0.28
	30–49	23.28±0.20	23.19±0.24	24.48±0.15	24.34±0.31
	50–59	—	—	(24.12±0.27)	(24.30±0.30)
	Average	23.50±0.08	23.30±0.10	24.46±0.07*	24.45±0.08*
Thoracic index	20–29	71.46±0.52	70.42±0.72	72.76±0.73	73.16±1.05
	30–49	72.88±0.76	74.24±0.70	74.50±0.61	76.10±0.92
	50–59	—	—	(76.58±1.11)	(77.91±1.05)
	Average	72.12±0.39	72.33±0.49	73.57±0.38*	74.60±0.40*

* Data for the age class 50–59 are excluded.

to be more variable; for example, the difference in mean chest breadth between male teachers and farmers (as shown in table 3) is slight and not significant but the difference in chest depth is statistically significant.

As sitting height decreases with age and thoracic contents do not decrease correspondingly, there must be an adjustment in the breadth and depth of the thorax. The ribs themselves and the thoracic girth increase little, if at all, and the increase is in depth rather than in breadth. The increased area of the thoracic cross section is achieved by the change from an ellipse towards a circle.

TABLE 4

Comparison of the Difference between Chest Depth and Breadth in Deep Respiration

AGE CLASSES	NUMBER MEASURED	IN DEEP INSPIRATION (cm) (I)	IN DEEP EXPIRATION (cm) (E)	DIFFERENCE (cm) (I—E)	$\frac{I-E}{\frac{1}{2}(I+E)}\times 100$
Chest Depth					
20–29	315	19.46	17.41	2.05	11.12
30–39	130	19.64	17.50	2.14	11.52
40–49	59	20.14	17.92	2.22	11.66
50–59	31	19.77	17.94	1.83	9.70
Average	535	19.59	17.53	2.06	10.92
Chest Breadth					
20–29	315	26.49	24.91	1.58	6.14
30–39	130	26.67	24.95	1.72	6.66
40–49	59	26.56	25.03	1.53	5.93
50–59	31	25.71	24.51	1.20	4.77
Average	535	26.47	24.91	1.56	6.07

N.B. Calculated from the data of DOHI on male prisoners (Essays of Anthropology, Human Genetics and Constitution, edited by Dr. Taniguchi, Vol. 14, 1949, Keiō Gizyuku University, Japan. In Japanese).

In Japan there are many people with marked spinal curvature which is not caused by any disease, but can be ascribed to the way of life and work—for instance, rice farming by the Japanese method, sewing and sitting on floor mats in the Japanese manner. Table 5 shows the correlation between senile curvature of the spine and roundness of the thorax. The spinal curve in the sagittal plane from the seventh cervical to the fourth lumbar vertebra was copied with a lead and plastic foc drawing curve ruler. The index of spinal curvature is expressed as the ratio of the length of the perpendicular (height of the curve) to the length of the cord. We calculated the coefficients of correlation between age, stature, thoracic index and index of spinal curvature on the basis of investigations of 58 senile persons housed in an asylum. The correlations of all of these factors are statistically significant except for age with thoracic index. The correlation between thoracic index and spinal curvature is especially marked and is demonstrable in the second order; that is, it is siginficant even when the influences of age and stature are excluded. In senile persons with markedly crooked spines the chest depth is sometimes greater than the chest breadth as in most quadrupedal mammalia. These extremely bent individuals walk with the aid of a stick holding the upper part of the body parallel to the ground in much the same manner as a quadruped. If the influence of the gravity of the thoracic contents is deduced to explain the form of the chest in quadrupeds, it should be considered in the case of such stooped senile individuals also.

TABLE 5

Correlation Coefficients among Age (1), Stature (2), Index of Spinal Curvature (3) and Thoracic Index (4) in Old People

$r_{12} = -.304$	$r_{13} = +.398$	$r_{14} = +.244$
$r_{12.3} = -.152$	$r_{13.2} = +.306$	$r_{14.2} = +.145$
$r_{12.4} = -.235$	$r_{13.4} = +.326$	$r_{14.3} = +.391$
$r_{12.34} = -.114$	$r_{13.24} = +.273$	$r_{14.23} = +.283$
$r_{23} = -.453$	$r_{24} = -.380$	$r_{34} = +.554$
$r_{23.1} = -.380$	$r_{24.1} = -.331$	$r_{34.1} = +.513$
$r_{23.4} = -.314$	$r_{24.3} = -.174$	$r_{34.2} = +.463$
$r_{23.14} = -.258$	$r_{24.13} = -.136$	$r_{34.12} = +.498$

SUMMARY

Studies of the thoracic form in 9,369 rural inhabitants of the Tōhoku area in Japan indicate certain developmental and retrogressive changes. The thorax of the newborn is rounder in transverse section but flattens increasingly until 4 years of age. In childhood, girls' chests are flatter than those of boys, but at the time of puberty, girls' chests suddenly become rounder and this is followed by a continuing tendency to greater roundness with age. The chests of boys continually become flatter until the 25–29 year old period. These changes seem to be developmental. Despite differences in the thoracic index between occupational groups, all such adult groups show a gradual rounding of the thorax after the age of 30 and the change accelerates with age. Two reasons have been considered as a cause of the rounding of the thorax with age: decrease in stature, especially in sitting height, and senile curvature of the spine.

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RELATIVE FAT PATTERNING: AN INDIVIDUAL CHARACTERISTIC *

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INTRODUCTION

ANTHROPOLOGISTS, clinicians and biometricians familiar with the appearance of nude subjects or experienced in viewing body-build photographs have long been impressed by marked individual differences in subcutaneous fat patterning. Such differences, equally apparent when soft-tissue radiographs are reviewed, do not always escape notice even when subjects are fully dressed. A number of seemingly different fat "patterns" may be intuitively identified in even a small series of persons of like sex, constant age, and from a single racial group.

The medical and anthropological literatures are well stocked with descriptive terms for different subcutaneous fat patterns. Without attempting to list all of them, and excluding pathological conditions like *adiposis dolorosa,* and such unusual depositions of fat as characterize *steatopygia,* certain patterns have been listed repeatedly. They include an "upper extremity pattern," a "girdle fat pattern," and a "lower extremity pattern." Variants in thoraco-abdominal fattening have been described as "D" shaped and "d" shaped. Further we read of a "juvenile fat pattern," an "adult fat pattern" and a "Rubens fat pattern" (Martin '28, Sheldon *et al.*, '40, Angel '49, Škerlj *et al.*, '53).

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While such designations are often devastatingly good, they lack quantification, depend much upon the judgment and preferred categories of the viewer, and may reflect in part configurations of the structures underlying the *tela adiposa* (*cf.* Škerlj *et al.*, '53, p. 593, Reynolds '51, p. 152). More important they may represent simple nutritional variants. As we have previously shown, the rate of fat deposition differs on different parts of the body so that (in absolute terms) the same individual may exhibit a different fat pattern when thin than he does when fat (Garn '54). Similarly, older and younger men may differ in the pattern formed by the absolute fat thicknesses not because of an age factor, but simply because the older man is fatter (Garn and Harper '55).

If, however, fat patterning is dealt with not in absolute terms, that is, the measured thicknesses themselves, but in terms of their z score transforms, pattern differences due solely to the amount of fat are eliminated. In this way it is possible to investigate interpersonal differences in fat patterning duly compensated for differences in the nutritive state. This technique of using z score profiles, which has had extensive use in psychometrics under the name of "profile analysis," seems highly applicable to the study of what we shall call *relative fat patterning*.[1]

It is the purpose of this study to investigate the extent of interindividual differences in "relative fat patterning." We shall indicate the degree to which individual males depart from the pattern expected for their degree of fatness. We shall demonstrate the existence of specific relative fat patterns in a series of adult males. And finally, we shall present evidence that individual relative fat patterns persist during weight loss, thus indicating that the relative fat pattern is an individual characteristic with some degree of permanence.

METHODS AND MATERIALS

The present study of relative fat patterning in the adult male is based on micrometer-caliper measurements of subcutaneous fat "shadows" as seen in soft-tissue X-rays of 94 adult males. The roentgenographic techniques and the location of the fat thicknesses measured are as described by Garn and Saalberg ('53), Garn ('54) and Garn and Harper ('55).

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[1] The z score is $(X-\bar{X})/\sigma$. In the present study the term "absolute fat pattern" as it applies to individuals refers to the pattern formed by the absolute or measured fat thicknesses, while the term "relative fat pattern" is used to designate the individual's z score profile. The absolute fat pattern may also be thought of as the "overt fat pattern," since it is this aspect of patterning that is perceived by the trained observer, while the relative fat pattern may be considered to be the "covert fat pattern," since it is ordinarily evident only after converting the absolute measurements into z scores.

Eighty-one of the subjects were adult male participants in the Fels Longitudinal Studies and were for the most part fathers of Fels children (Garn '54). Thirteen of the men were subjects in an investigation of controlled weight loss, conducted at the Laboratory of Physiological Hygiene, University of Minnesota (Brožek *et al.*, '55). These latter men were X-rayed by the author and Dr. Josef Brožek during a maintenance period and again at the end of a period of restricted caloric intake, employing the same roentgenographic procedures used on the larger series.

Each of the 9 measured fat thicknesses for each subject was converted into the corresponding z score or standard score value (see Guilford, '36) using the normative data published earlier (Garn '54).[2] In this way the 9 fat measurements, which differ markedly in thickness and absolute variability, were placed upon a comparable basis. Z scores, it may be recalled, have a mean of 0 and a standard deviation of *1* (Guilford '36, p. 86, Edwards, '47, p. 47); thus a hypothetical individual exactly 1 sigma above the mean in all 9 fat measurements would be represented by a z score value of $+1$ throughout. The appearance of such z score profiles is depicted in figure 1.

The extent of variability within an individual was measured by the statistic σz. This is simply the standard deviation of the individual's own z scores for subcutaneous fat. For the hypothetical individual described above σz would be 0 since all of his z scores were identical. Individuals more variable in relative fat patterning might have σz values of 0.5, 1.0 or even 1.5. σz therefore measures the extent of individual variability in subcutaneous fat patterning, corrected for the amount of fat present.

However, while σz provides a numerical measure of relative fat variability, it cannot of itself indicate where an individual departed from the general pattern. Accordingly, those individuals with above-median

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[2] The use of z scores implies either that the distributions are normal or of the same form. This is not strictly true, in the present case, since χ^2 tests of the 36 combinations reveal a significant degree of heterogeneity of the distributions, with the distribution of hip fat somewhat different from 5 of the 8 others. However, this does not affect the general nature of the findings and especially the pattern comparisons. For a short discussion and further references see McNemar ('49, p. 38).

σz values were hand-sorted, and their z score values plotted on suitable coordinate paper, as shown in the lower part of figure 1. In this way it was possible to analyze, and in some cases categorize the diverse relative fat patterns making for high σz values. The "pattern index" technique developed by Lacey and Van Lehn ('52) was followed in part.

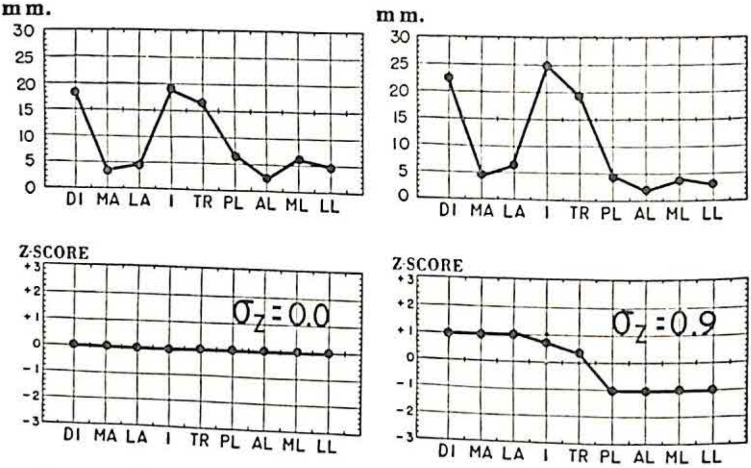

FIG. 1. Comparison of absolute fat patterns (above) and corresponding relative fat patterns (below). Left, hypothetical individual exactly at the mean in all 9 fat thicknesses. His mean z and fat variability score (σz) are both 0. Right, hypothetical individual with relatively thick arm fat and relatively thin leg fat. Though the difference between the two men is evident in the absolute fat patterns, it is striking in the relative patterns and in the fat variability scores.

The abbreviations: DI, MA, LA, I, TR, PL, AL, ML, and LL refer to deltoid insertion fat, medial arm fat, lateral arm fat, iliac fat, trochanteric fat, posterior leg fat, anterior leg fat, medial leg fat and lateral leg fat.

Finally the permanence of such z score fat patterns, or more precisely their resistance to change during weight loss, was investigated in the Minnesota group. Here the z score profile patterns before and after weight loss for each individual were plotted and compared. The degree of similarity or fit between the "before" and "after" patterns was expressed numerically as σdz, the standard deviation of the z score differences. Like σz, σdz also has a theoretical lower limit of 0, and no upper limit. A σdz of 0 would be attained if an individual in losing fat lost the same amount (in z score units) in all 9 fat thicknesses measured. Thus σdz measures the degree of *similarity* between z score profile patterns, and may be called the *profile similarity index.*

These procedures which deal with z scores, rather than absolute fat thicknesses, are made necessary by the facts that the different fat thicknesses measured differ considerably in their mean values, and that the rate of fat deposition or withdrawal is non-uniform over different parts of the body.

FINDINGS

1. *Relative fat variability scores* (σz)

To begin with, the standard deviation of individual z scores for subcutaneous fat (σz) was calculated for each of the 81 males in the basic series. As shown in table 1, the value of σz ranges from a low of 0.26 to a high of 1.45. The lowest value observed approaches the theoretical minimum of 0.0, and indicates a high degree of z score uniformity, and an almost total absence of relative fat patterning. The high values, on the other hand, are indicative of considerable internal variability, disproportion or "dysplasia" in subcutaneous fat deposition. Since σz is a new statistic in this work, it is of interest to note that the mean was 0.61, and the distribution considerably skewed, with a Pearsonian coefficient of skewness (Sk) of 0.75. While such skewness is to be expected where fat is concerned, it indicates that the median rather than the mean σz should be employed as a measure of central tendency. Accordingly, in selecting individuals with high σz values, for the second

TABLE 1

Distribution of Relative Fat Variability Score (σz) *for 81 Adult Men*

RANGE	NO.	PERCENT	CUMULATIVE PERCENTAGE
0.00-0.19	0	0	0
0.20-0.39	11	14	14
0.40-0.59	37	46	60
0.60-0.79	18	22	82
0.80-0.99	10	12	94
1.00-1.19	2	2	96
1.20-1.39	1	1	97
1.40-1.59	2	2	99
1.60-1.79	0	0	99

The mean is 0.61 and the median is 0.56.

part of the study, selection was based on above-median rather than above-average σz scores.

Though, anthroposcopically, older men seem to be more variable in absolute fat patterning, there was no evidence that age alone influenced the relative fat variability scores. The mean σz for 40 younger men did not differ significantly from the mean for 41 older men. As a confirmation the product moment r between age and σz failed to attain significance at the 5% level. In like fashion, thin and fat men did not differ significantly in mean σz (table 2). However, a low, but positive and significant correlation of 0.32 between trochanteric fat and σz suggested at least that fatter, or fatter-hipped, men may be slightly more variable in relative fat patterning. Under these circumstances, some degree of caution may be required, before saying that the very fat are more "dysplastic" than the very thin.

TABLE 2

Fat Variability Scores (σz) for Older and Younger and Fatter and Thinner Men

GROUP	NO.	MEAN AGE	σz RANGE	MEAN σz	S. D.
Total group	81	39.8	0.26-1.45	0.61	0.24
Younger men	38	30.4	0.26-1.08	0.62	0.21
Older men	43	48.1	0.32-1.45	0.61 [3]	0.26
Leaner men [1]	41	39.5	0.28-1.45	0.57	0.25
Fatter men [2]	37	40.8	0.32-1.43	0.66 [3]	0.25

[1] Trochanteric fat below 16.6 mm.

[2] Trochanteric fat over 16.6 mm.

[3] Differences between means not significant at 5% level.

2. *Relative fat patterns*

Since a given value of σz merely expresses the degree of variability in the individual's z scores for fat, without explaining the source of the variability, it was of interest to explore the various relative fat patterns in order to determine whether there were "upper extremity," "lower extremity" and "girdle fat patterns," etc. Therefore the z score values for subcutaneous fat were plotted for all men above the median σz, and the z score profile patterns were examined further.

Of the 40 men so plotted, it was not possible to describe or "name" the pattern anatomically in 23 cases. That is, these 23 men high in σz were by inspection high or low in individual fat thicknesses (medial arm, posterior leg, etc.) or showed highly localized highs and lows. In 17 men, however, the patterns involved anatomical regions, with deviations up to two z scores from their own mean z and ranges up to 4 z scores.

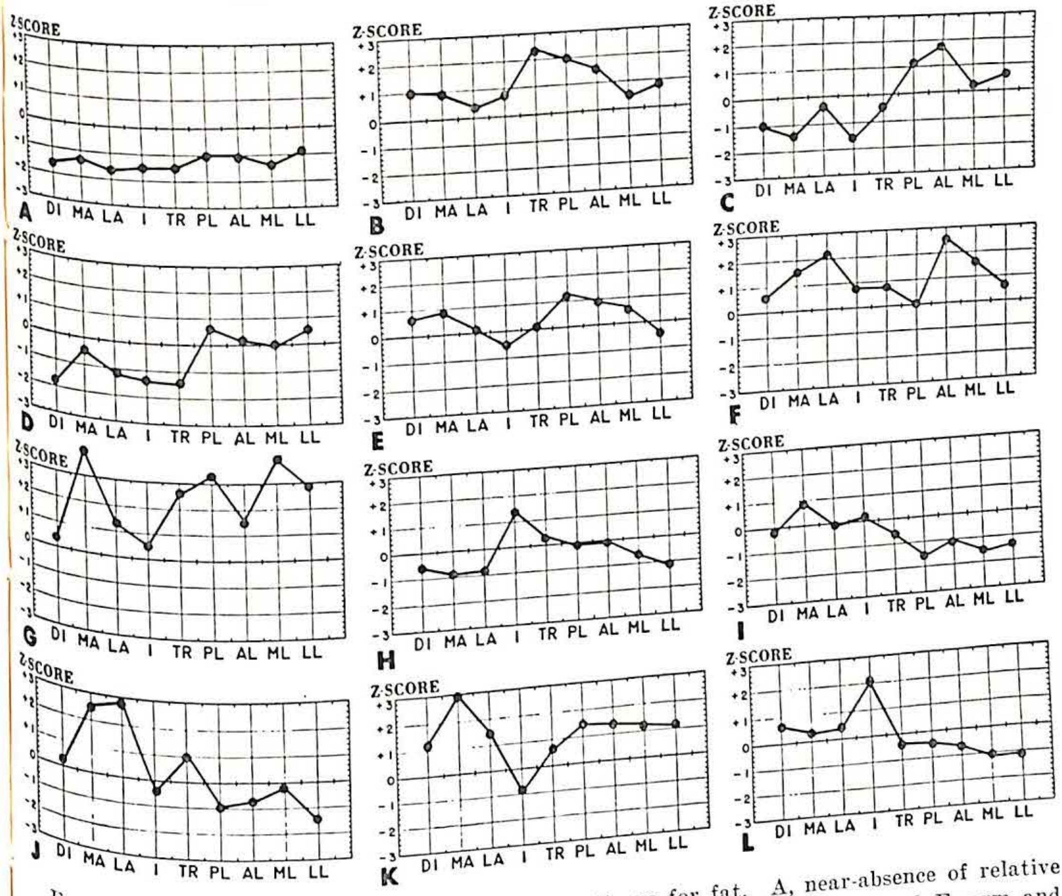

FIG. 2. Twelve representative z score profile patterns for fat. A, near-absence of relative fat patterning. B, lower segment pattern. C and D, leg fat patterns. E and F, arm and leg patterns. G, example of a pattern not easily categorized. H, iliac fat pattern. I, arm fat pattern. J, arm-trochanter fat pattern. K, low hip-fat pattern (— 2σ below the individual's own mean). L, high hip pattern (1.9σ above individual's own mean). The abbreviation DI, MA, LA, etc. refer to specific fat thicknesses as in figure 1.

Such cases where lower leg fat or arm fat responded as a whole were simpler to name.

In 5 men there was a clearly-marked leg fat pattern characterized by elevated z score values for the entire leg region. Examples are shown in figure 2, c and d, wherein it is evident that a leg fat pattern (averaging two or more z scores above the individual's average z) may be found both in men who are thin and those others who are fat. Three additional men showed a combined arm and leg pattern, giving their z score profiles a notably sinusoidal appearance. One man, out of the 40, evidenced a combined pelvis and leg pattern, a combination rarely to be expected on a chance basis alone.

In all, 3 men exhibited the kind of concomitant trochanteric-iliac fat excess, that could be called a "girdle fat pattern." However, it should be noted that the z scores correct for the usual tendency to deposit fat differentially in this region, so that these 3 men are exceptionally "hippy" specimens. The individual pictured in figure 2, h, is average in his total fat thickness but markedly deviant in iliac fat.

Other nameable patterns include an arm-hip pattern (figure 2, i), a true arm pattern (figure 2, j), and a pattern involving low iliac fat (figure 2, k). The individuals shown in figure 2 k and l are quite exceptional in that their iliac fat is nearly 2 standard scores out of line from their *own* z score mean.

Therefore it is evident that there are a number of relative fat patterns contributing to an above average σz, and characteristic of individuals. While some of these relative fat patterns can be described by terms previously used (lower-extremity, upper-extremity etc.) over half of them are not easily described in words. Under these circumstances the z score profiles, or better, t score profiles, may prove more useful in their original form than when further categorized. To put it differently, there are more, and more complicated, relative fat patterns than we can conveniently force into categories.

3. *Permanence of the relative fat pattern*

While it is evident that there are marked interpersonal differences in relative fat patterning, one may well ask whether any given relative fat pattern possesses permanence, and persists despite changes in nutritional status. Fortunately data on the 13 young men from the weight loss series allow us to answer this question with some degree of assurance.

As shown in figure 3, 1 through 13, the individual z score profile patterns after weight loss closely resemble the initial patterns and in several cases are practically identical (figure 3, 1, 2, 3, and 12). In none of the 13 cases did food restriction cause a reversal of pattern or even a marked change. Interestingly, the tendency for the individual relative fat patterns to hold, despite generalized loss of fat, is as marked in those men who were initally obese as among those who were lean even before food restriction.

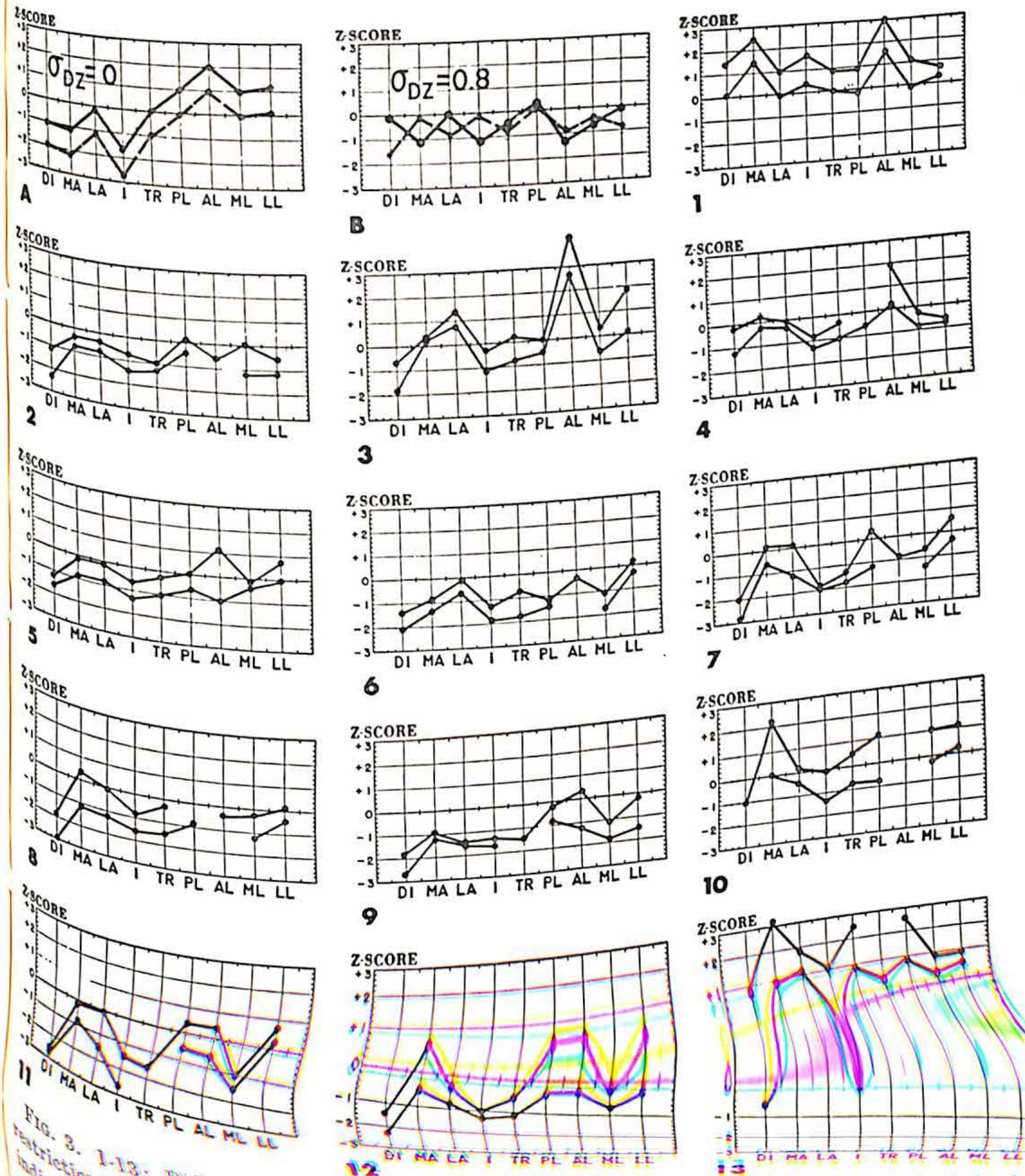

FIG. 3. 1-13; Relative fat patterns of 13 young men before and after a period of food restriction. Despite generalized fat loss, as shown by the lower level of each "after" pattern, individual pattern profiles maintained their identity.

A and B; hypothetical patterns illustrating (A) perfect pattern congruence and (B) pattern dissimilarity with several reversals.

However, while the "before" and "after" z score profiles provide striking visual proof of the resistance of the relative fat pattern to changed nutritional status, the plotted patterns do not afford a quantitative measure of pattern similarity. Therefore, quantification was accomplished by computing σdz which is the standard deviation of the differences expressed in z score units. As pointed out previously, and as illustrated in figure 3, A and B, if two z score profile patterns are identical, the value of σdz will be 0. σdz, therefore, measures the degree of *profile similarity.*

As shown in table 3, the observed values of σdz (the profile similarity index) range from 0.20 to 0.50 for the 13 men in the weight reduction series, with a mean of 0.39 ± .03 for the group as a whole. In contrast the values of σdz for 13 randomly selected pairs of men were much higher, ranging from 0.50 to 1.36 and the mean σdz for these randomly selected pairs was 0.77 ± .07. Thus an individual continues to resemble himself in the relative fat pattern, despite considerable and rapid weight loss.

Since all of the men in the weight-loss series had lost fat during the course of the experiment, there were possible objections to the use of random pairs for comparison, even though the actual pairings were made by a disinterested member of the Fels Institute staff. Accordingly, a second set of pairs was set up, matched on a weight basis to the starvation series men before and after food restriction. Here, the values of σdz ranged from 0.41 to 1.47, with a mean of 0.67 ± .07. These results were substantially similar to those mentioned above.

These particular findings may be summarized by saying that individual relative fat patterns hold their shape during weight reduction even though the measured fat thicknesses (*i. e.,* the absolute fat patterns) change. The use of σdz makes it possible to demonstrate that the relative fat patterns of an individual under two different conditions of nutriture are more alike than the relative fat patterns of two different individuals, whether selected on a weight basis or not.

DISCUSSION

In this communication we have returned to a consideration of the individual, the prime unit of constitutional research, though limiting ourselves to the study of individual differences in one tissue layer (the *tela adiposa*), one sex and age group (adult males), and to purely objective measurements. We have shown that it is possible to go beyond

TABLE 3

Profile similarity indices for weight-reduction series, random pairs and weight matched pairs

Subject or Pair Number	I WEIGHT-LOSS SERIES		II RANDOMLY-MATCHED PAIRS		III WEIGHT-MATCHED PAIRS[1]	
	Weight loss Kg	σdz	Weight difference Kg	σdz	Weight difference Kg	σdz
1.	14.4	0.34	—12.1	0.69	7.6	0.79
2.	9.4	0.42	5.5	0.62	10.0	0.67
3.	10.2	0.48	—13.2	1.36	10.4	0.62
4.	8.9	0.48	—0.9	0.63	8.5	0.76
5.	9.8	0.28	—6.6	0.73	8.8	0.68
6.	8.3	0.20	—13.3	0.57	7.9	1.47
7.	8.9	0.43	—26.8	0.55	9.5	0.47
8.	10.0	0.30	6.9	0.76	10.0	0.66
9.	6.9	0.48	—7.9	0.50	6.7	0.81
10.	9.4	0.53	3.9	0.61	9.5	0.47
11.	5.0	0.39	0.8	0.93	8.8	0.41
12.	11.3	0.24	—1.4	1.01	9.8	0.65
13.	6.9	0.50	26.0	1.09	15.0	0.51
	9.2 ± .64	0.39 ± .03	—3.01 ± 3.51	0.77 ± .07	9.4 ± .54	0.69 ± .07

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[1] Matched to initial and terminal weights of group I.

the absolute fat patterns, which are highly responsive to changes in caloric intake, to the relative fat patterns, which appear to be of a more permanent nature.

The measure σz, the standard deviation of the individual's own z scores for fat, provides a direct measurement of relative fat variability. σz, which has a lower limit of 0, and no upper limit, measures the extent to which the individual departs from a straightline z score profile. Since z score units are used, and variability is measured about the individual's own mean z, age does not enter into the picture, and for the most part differences in the degree of obesity may be ignored. A male with a σz greater than 0.61, or better 0.90, may be considered dysplastic (in regard to distribution of fat) whether fat or lean.

While a high σz indicates that an individual does have an unusual fat pattern, it cannot of itself indicate why or where. Here, actually plotting the z score profiles for fat becomes a necessity. As shown by the analysis of individual z score profiles, there are a number of distinct and individually characteristic relative fat profiles. Some of them can be categorized as "arm-fat," "leg-fat," "arm and leg fat," etc. On the other hand, there are a larger number, quite distinct in the profiles, that cannot be categorized, or categorized in simple language. And, since our interests extend well beyond the level of simple description there is no need to do so.

The probability that a given relative fat pattern is an individual characteristic, possessing some degree of permanence through time, is heightened by the findings in the weight reduction group. Here all of the men lost weight (up to 14 kg) and all lost fat in all 9 fat layers measured. Consequently the z score patterns were all displaced downward at the end of the weight loss period. Yet the individual patterns tended to "hold." The extent of similarity between the individual "before" and "after" patterns was striking, and there was little difficulty in recognizing each individual from his post-starvation z score pattern. However, the permanence of such patterns has not yet been demonstrated over longer time periods, or under different nutritional regimens.

Still, it is important to point out the utility of the relative fat pattern concept. As is well known, absolute fat patterns undergo qualitative changes during adolescence and less striking quantitative changes thereafter. Under these circumstances, permanence of type cannot be sought either in the absolute fat thicknesses, or in the patterns formed by the absolute fat thicknesses. Relative fat patterning seems to afford the only approach both to the problem of permanence of type and the problem of genetic determinants, as far as the *tela adiposa* is concerned.

Previous workers have demonstrated a general tendency for children to hold position, either on a rank basis or a percentile basis, in the thickness of specific fat layers during the growing period (Stuart *et al.*, '40, Stuart and Sobel '46, Reynolds '51). While such constancy suggests a genetic determinant, it is equally likely that children maintain their eating habits over the years: a juvenile trencherman continuing to be a trencherman and a child with a bird-like appetite continuing his avian behavior. Reynolds ('51) further demonstrated the existence of sibling similarities in the thickness of the *tela adiposa*. Again, while the possibility of a genetic determinant cannot be ignored, the fact that siblings share a common diet should not be discounted either. Under these circumstances the relative fat pattern may prove of maximum use both in studying constancy of type and in searching for genetically-determined variants in the *tela subcutanea*.

Finally, it should be pointed out that the relative fat pattern does not replace the absolute fat pattern. The absolute fat pattern comes closest to what we perceive. The absolute fat pattern is affected by age, sex, and nutritional status, and it is more than likely that the absolute fat patterns of different races differ to an important degree. The relative fat pattern, on the other hand, is much more of an individual characteristic and, in adulthood at least, is seemingly much more resistant to change. Both aspects of fat patterning are important adjuncts to research on the subcutaneous fat. Absolute fat patterns obviously relate to nutritional studies. Relative fat patterns, on the other hand, afford an opportunity for the investigation of the individual and are far more likely to find their origin in the germ plasm than in the "groaning board."

SUMMARY

1. Individual differences in *relative fat patterning* were investigated after converting absolute measurements into their corresponding z transforms. In this way patterned differences due solely to the amount of subcutaneous fat were eliminated.

2. The measure σz (the standard deviation of the individual's own z scores for fat) was used to indicate the extent of *relative fat variability*.

In 81 adult males σz ranged from 0.29 to 1.45 with a mean of 0.61. It was suggested that σz could serve as an objective measure of fat "dysplasia."

3. As tested in various ways, σz was not affected by age and only slightly, if at all, by the total amount of fat.

4. Individual relative fat patterns were investigated in the z score plots of 40 men with above-median σz values. A number of characteristic relative fat patterns were found, some of them resembling patterns described in the literature. In 17 men there were distinct and nameable relative fat patterns. However, in 23 cases, the relative fat patterns were not susceptible to simple naming.

5. The possibility that such relative fat patterns are permanent and resistant to changes in nutritional status was investigated by comparing the z score profile patterns of 13 young men during a period of food restriction.

6. Visually, the tendency of an individual to maintain his z score profile pattern was striking. Using a quantitative measure of pattern similarity (σdz) this impression was confirmed.

7. It was observed that the relative fat pattern is an individual characteristic, having some permanence and resistance to change under nutritional stress, and that the relative fat pattern (in contrast to the absolute fat pattern) may be genetically determined.

ACKNOWLEDGMENTS

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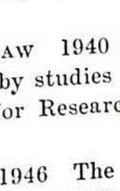

A MARKOV PROCESS MODEL FOR FOLLOW-UP STUDIES

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1. INTRODUCTION

THE purpose of this paper is to introduce the general, time stationary, continuous, Markov process with a finite number of states, as a model for the follow-up study of a disease. A special case of such a process was introduced by Fix and Neyman ('51) as a model for the study of cancer or other disease.

In models used up to the present time cure rates or death rates have generally been used to measure the effectiveness of a treatment. These rates may be too coarse in that they overlook other important characteristics of the disease or treatment. They may also be not quite relevant in that the disease may cause death only indirectly and only after many years, or else the treatment does not cure in the sense of the patient becoming free of at least the gross symptoms, but only retards the progress of the disease. This is the situation with many, if not most, of the degenerative diseases. Under these circumstances, a medical scientist will most likely incorporate intuitive and non-quantitative measures into his appraisal of the treatment.

To introduce other quantitative measures of a treatment, it is necessary to consider more elaborate models of the temporal progress of the disease than have hitherto been used. One such elaboration, where it is appropriate to do so, is to define several states that a patient may be in. For example, in studying arthritis we might define the following states: 1, active arthritis; 2, quiescent; 3, death from arthritis; 4, lost from observation or death from other causes. This might be enlarged by replacing state 1 with several states representing a range of activity from active to quiescent. The exact number of states would depend on the purposes of the investigation and the ability of the investigator to discriminate between the states. Given such a set of states the patient who enters the study is regarded as traversing a path: he enters the study in some initial state and from time to time transfers from one state to another and eventually leaves the study in some final state. It is assumed that the paths are determined by a set of stochastic or probabilistic laws, *i.e.*, the laws specify the probability of any particular path, though not the path itself. With the aid of such a model the disease process is studied as a stochastic process. The new measures which it is hoped are useful are derived from certain characteristics of the stochastic process. One such characteristic is the probability that a patient who enters the study classified in one particular state will, after T years, leave the study in some other, or the same, particular state. Such a T year transition probability, as it is called, is a generalization of the more familiar T year survival rate. If there are k states, then there are k^2 such T year transition probabilities. As a second characteristic we have, for all patients who enter the study in one particular state and leave the study after T years, the average time spent in each of the states. As an illustration of this second characteristic, suppose a drug is being tested for its power of inhibiting the activity of arthritis. Then the average length of time which a person spends in a less active state of arthritis, who originally received treatment in a more active state, might be a measure of the effectiveness of the drug.

The chief statistical problem is to estimate the set of parameters which defines the Markov process, by means of a sample of paths. The sampling scheme considered in this paper is one which consists of periodic observations on patients, that is, if a patient enters the study at time t_0, he is observed at times $t_0 + t, t_0 + 2t, t_0 + 3t$, etc., and only the information concerning the state of the patient at these times is used to estimate the parameters of the process.

In section 2 a brief description of the Markov process used as a model is given. In section 3 certain useful characteristics of the process are defined. These sections are included for the sake of completeness only. A more thorough treatment of the mathematical aspects of the model is given, for example, by Doob ('53), or Feller ('50) both of whom give extensive bibliographies. In section 4, the estimates of the parameters (called transition rates) of the model are given. Estimates of the characteristics are then derived from the estimates of the transition rates. Section 5 treats of the large sample distribution of the estimates, and the next two sections, 6 and 7, of tests of hypotheses and tests of goodness of fit of the model, respectively. In section 8 the model is generalized to two contiguous study periods. In section 9 an example is given.

The terminology used in this paper refers to disease processes. As far as the theory is concerned, this is only a matter of convenience and is not meant to imply any limitation to the study of disease alone.

2. A BRIEF DESCRIPTION OF THE MODEL

We assume that during the course of a follow-up study a patient is at every instant of time in one and only one of k states which we label $S_1, S_2, \cdots, S_k$. The follow-up study is of length T, which means that each patient is under observation for a period of time T following his entry into the study. The actual starting times of the patients are ignored and the entry times all set equal to zero. The total period of the study is therefore from $t = 0$ to $t = T$, and the fact that a patient is assumed to be in one of the k states at each instant of this interval of time makes the process, by definition, a continuous one.

The phrase, under observation, in the context of this paper, does not mean strictly physical observation; it means rather, counted as being in the study and capable of being classified in one of the k states at any instant of time, while in the study. By this definition, for example, a patient who dies during the study remains formally under observation until his time T has elapsed, if death is one of the states. We remark also that the times $t = 0$ to $t = T$ need not, in practice, represent the actual length of the follow-up study. These two times may, for example, represent years 0 to 5, or 6 to 10, or any interval of a 20 year follow-up study.

We are concerned with certain events called transitions. A transition is an event of the following form: a patient at time t_1 is in state S_i and at time $t_2 > t_1$ is in state S_j, where i and j may be equal or unequal. Nothing is said about the state of the patient at any other time. Associated with these events are probabilities. If a patient is in state S_i at any time t_0 we assume that there exists a conditional probability $p_{ij}(t)$ that at a later time $t_0 + t$ the patient will be in state S_j, for $i, j = 1, \cdots, k$ and $0 \leq t_0 \leq t_0 + t \leq T$. The probabilities $p_{ij}(t)$ are called transition probabilities. We have defined the transition probability to depend only on the difference $(t_0 + t) - t_0 = t$ and not on the actual time points t_0 and $t_0 + t$; by definition this is the property of stationarity. When making statements about $p_{ij}(t)$ for all values of t

in the interval $[0, T]$ we will also refer to $p_{ij}(t)$ as a transition probability, in order not to complicate the terminology too much, though strictly speaking, $p_{ij}(t)$ should then be called a transition probability function.

The additional assumptions that characterize our model are as follows. When $t=0$,

$$p_{ij}(0)=\begin{cases}1 & \text{if } i=j\\ 0 & \text{if } i\neq j\end{cases}. \tag{2.1}$$

The transition probabilities are also continuous with respect to t at $t=0$, *i. e.*,

$$\lim_{t\to 0} p_{ij}(t)=\begin{cases}1 & i=j\\ 0 & i\neq j\end{cases}. \tag{2.2}$$

The last assumption is that our process has the Markov property. Loosely speaking this means that the conditional probability of the transition from S_i to S_j in time t does not depend on the history of the patient previous to the time he was in state S_i. More precisely, if $p_i(0)$, $i=1,\cdots,k$ is the probability of being in state S_i at $t=0$, and $0<t_1<t_2<t_3<\cdots<t_n<T$ are any finite set of time points, then the probability that a patient is in state S_{i_0} at $t=0$, S_{i_1} at $t=t_1,\cdots, S_{i_n}$ at $t=t_n$ is

$$p_{i_0}(0)\,p_{i_0i_1}(t_1)\,p_{i_1i_2}(t_2-t_1)\cdots p_{i_{n-1}i_n}(t_n-t_{n-1}).$$

The set of transition probabilities $p_{ij}(t)$ are conveniently assembled into a transition matrix $P(t)=\{p_{ij}(t)\}$. We observe that when $t=0$, $P(0)$ is the identity matrix. Matrix terminology is very convenient for our presentation and it will be used extensively.

It can be shown (Doob '53, pp. 238-243) that there exists a unique matrix Q such that for $0\leq t\leq T$,

$$\begin{aligned}P(t)&=I+Qt+Q^2\frac{t^2}{2!}+Q^3\frac{t^3}{3!}+\cdots\\ &=e^{Qt}\end{aligned} \tag{2.3}$$

symbolically. Q does not depend on t; its elements are the instantaneous transition rates, as may be seen from the relation

$$\lim_{t\to 0}\frac{1}{t}(P(t)-I)=Q.$$

The elements of Q will therefore be called transition rates and Q the transition rate matrix, or simply rate matrix. Q together with the initial states of the patients determines the process, so the primary estimation problem is to estimate Q.

The transition rates have the following properties:

$$q_{ij}\begin{cases}\geq 0 & i\neq j\\ \leq 0 & i=j\end{cases} \tag{2.4}$$

$$\sum_{j=1}^{k} q_{ij}=0 \qquad i=1,\cdots,k. \tag{2.5}$$

Any square matrix Q with the properties (2.4) and (2.5) is the rate matrix of some process (of the type we are concerned with). This is a useful property enabling us to manufacture processes at will.

In conclusion, the most important conditions which the disease process must satisfy, at least approximately, in order for the model to apply, are the following:

1. During the period $[0,T]$ of the study, the patient is at any time in just one of a finite number of clearly defined states, $S_1,\cdots S_k$.

2. The probability that a patient in state S_i $(i=1,\cdots,k)$ at any time during the study, is subsequently in state S_j $(j=1,\cdots,k)$ at a later time in the study, depends on the two states in question but not on the previous history of the patient.

3. The probability that a patient in state S_i at time t_1 is in state S_j at a later time t_2 depends on the difference t_2-t_1, but not on the initial time t_1.

3. CHARACTERISTICS OF THE DISEASE PROCESS

Starting with a given rate matrix Q, which may be an estimate or else theoretically given, we can derive certain characteristics of the process.

a. From (2.3) we can determine the transition probabilities for any time interval, in particular for the total period $[0,T]$, by letting $t=T$.

b. It may sometimes be desirable to find the transition probabilities corresponding, not to the given Q, but to a rate matrix obtained from Q by changing the values of some of the elements of Q, while at the same time satisfying the restrictions (2.4) and (2.5). As an important particular case, suppose the state S_δ is the state of being lost from observation. The rates $q_{i\delta}$ $(i=1,\cdots,k;i\neq\delta)$, representing rates of transfer from the other states to S_δ, will affect the transition probabilities in a way which, we usually assume, has nothing to do with the disease.

By substituting 0 for these $q_{i\delta}$'s and altering the diagonal elements of Q to satisfy (2.5), a new transition matrix can be calculated for the hypothetical situation in which no patients are lost from observation. In general, if the transition rates $q_{\alpha\beta}, q_{\gamma\delta}, \cdots, q_{\mu\nu}$ have been suppressed in this way, then the altered rate matrix will be called the net rate matrix and written $Q_{\alpha\beta|\gamma\delta|\cdots|\mu\nu}$, and its elements called net transition rates. The corresponding transition matrix will be called the net transition matrix and written $P_{\alpha\beta|\gamma\delta|\cdots|\mu\nu}(t)$, and its elements called net transition probabilities.

c. From a given rate matrix another characteristic that can be derived is: for each pair of states S_i, S_j, the expected time spent in state S_j during the period $[0, T_0]$, $0 \leq T_0 \leq T$, for a patient who is initially in state S_i. This expectation will be noted by $e_{ij}(T_0)$; it is given by

$$e_{ij}(T_0) = \int_0^{T_0} p_{ij}(t)\,dt$$

where $p_{ij}(t)$ is determined by Q from (2.3). The integral is the limit as $N \rightarrow \infty$ and $\| t_n - t_{n-1} \| \rightarrow 0$ of the approximating sum

$$\sum_{n=1}^{N} p_{ij}(t_{n-1})\,p_{ij}(t_n - t_{n-1}) \cdot (t_n - t_{n-1})$$

where $0 = t_0 < t_1 < \cdots < t_n = T_0$.

The matrix of expectations

$$e(T_0) = \{e_{ij}(T_0)\} \qquad i, j = 1, \cdots, k$$

may be computed using (2.3). We have

$$\begin{aligned} e(T_0) &= \int_0^{T_0} P(t)\,dt \\ (3.1) \qquad &= \int_0^{T_0} I\,dt + Q\int_0^{T_0} t\,dt + Q^2 \int_0^{T_0} \frac{t^2}{2!}\,dt + \cdots \\ &= IT_0 + Q\frac{T_0^{\,2}}{2!} + Q^2\frac{T_0^{\,3}}{3!} + \cdots. \end{aligned}$$

The matrix $e(T_0)$ will be called the residence matrix and its elements, residencies. If $Q = Q_{\alpha\beta|\cdots|\mu\nu}$ is substituted in (3.1), then the resulting residence matrix will be called the net residence matrix and written $e_{\alpha\beta|\cdots|\mu\nu}(T_0)$. Its elements will be called net residencies.

It is clear from the above that the transition and residence matrices depend entirely on Q. It is convenient therefore to estimate Q first and then to estimate the transition and residence matrices using the relations (2.3) and (3.1).

4. ESTIMATION IN THE MODEL

The estimate of Q is based on a series expansion of Q in terms of $P(t)$. Writing $P^*(t) = P(t) - I$, (2.3) becomes

$$I + P^*(t) = e^{Qt}. \tag{4.1}$$

Proceeding heuristically we then write

$$Qt = \log(I + P^*(t)) = P^*(t) - \frac{(P^*(t))^2}{2} + \frac{(P^*(t))^3}{3} - \cdots. \tag{4.2}$$

In appendix 1 it is proved that (4.1) and (4.2) are equivalent provided the series in (4.2) converges elementwise. A necessary and sufficient condition that the series converges is that the roots of $P^*(t)$ are all less than one in absolute value (Wedderburn, '34). This condition is not very convenient to apply in practice as it may involve considerable computation. For our purposes the following condition is more useful. If

$$p_{ii}(t) > \tfrac{1}{2} \text{ for } i = 1, \cdots, k \tag{4.4}$$

then the series converges. The proof is given in appendix 2.

Let us suppose now that at periodic intervals t' of time, starting from their respective times of entry into the study, patients are classified into one of the states $S_1, \cdots, S_k$. If a particular patient is classified s times in this way, his observed path will consist of the sequence of states $S_{i_0}, S_{i_1}, \cdots, S_{i_s}$, where S_{i_0} is the initial state, S_{i_1} the state after time t' has elapsed, etc. The likelihood of this path is

$$\pi_{i_0} p_{i_0 i_1}(t') p_{i_1 i_2}(t') \cdots p_{i_{s-1} i_s}(t')$$

where π_{i_0} is the probability of the patient entering the study in state S_{i_0}, while the remaining probabilities correspond to the succeeding transitions. Assuming that patients behave independently, the likelihood of the observed paths of n patients, after rearranging, is therefore

$$L = \prod_{i=1}^{k} \pi_i^{n_i} \prod_{i,j=1}^{k} p_{ij}(t')^{n_{ij}} \tag{4.5}$$

where n_i is the number of patients entering in state S_i, n_{ij} is the total number of transitions $S_i \to S_j$ and $n = n_1 + \cdots + n_k$. In order that the likelihood not vanish we define $\pi_i^{n_i} = 1$ if $n_i = 0$ and similarly we define $p_{ij}(t')^{n_{ij}} = 1$ if $n_{ij} = 0$. A straightforward computation yields the maximum likelihood estimate of $p_{ij}(t')$:

$$\hat{p}_{ij}(t') = \frac{n_{ij}}{n_{i1} + \cdots + n_{ik}} \qquad i, j = 1, \cdots, k. \tag{4.6}$$

We observe that the π_i's do not enter, so that the estimating procedure is independent of the particular scheme of entry of the patients. We also observe that in order for $\hat{p}_{ij}(t')$ to be defined, the denominator in (4.6) must not vanish. The interpretation of this fact is that somewhere in the course of the follow-up study patients must be observed to be in each of the designated states.

In estimating Q we consider first the case that there are no *a priori* restrictions on the elements of Q, and second that there are restrictions of the form $q_{ij} = 0$ for certain of the transition rates. These restrictions would occur when it is known that the corresponding transitions $S_i \to S_j$ cannot occur.

Case 1. No *a priori* restrictions on the rates. Letting

$$P(t') = \{\hat{p}_{ij}(t')\} \quad \text{and} \quad \hat{P}^*(t') = \hat{P}(t') - I,$$

the estimate of Q is taken to be

$$Q = \frac{1}{t'}\,[\hat{P}^*(t') - \tfrac{1}{2}(\hat{P}^*(t'))^2 + \tfrac{1}{3}(\hat{P}^*(t'))^3 - \cdots] \tag{4.7}$$

provided the series converges. If the series converges, $\hat{Q}$ is the unique maximum likelihood estimates of Q. It is not certain, however, that the series will converge even if the roots of $P^*(t')$ are less than one in absolute value, since $\hat{P}^*(t')$ is a sample matrix and some of its roots may, by chance, exceed one. However, the probability of the series not converging can be made arbitrarily small by choosing t' sufficiently small. Condition (2.2) states that the parameters $p_{ii}(t')$ converge to 1 as $t' \to 0$, and the $\hat{p}_{ii}(t')$'s, being maximum likelihood estimates of the $p_{ii}(t')$'s, converge in probability to the latter. Hence, a practical solution is to choose t' so that the $p_{ii}(t')$'s are (sufficiently) greater that $\frac{1}{2}$. This will make the probability that any of the $\hat{p}_{ii}(t')$'s exceed $\frac{1}{2}$ (and the series possibly not converging thereby) small. Some *a priori* information as to the magnitudes of the $p_{ii}(t)$'s as functions of t is needed in order to choose t' suitably. In this paper we will assume that the series in (4.7) does converge.

Case 2. Some of the parameters q_{ij} are known to be zero. In this case we take as our estimate of Q a modification of $\hat{Q}$. First $\hat{Q}$ is computed by (4.7). Then if $q_{\alpha\beta}$ is known to be 0, $\hat{q}_{\alpha\beta}$ is replaced by 0 and $\hat{q}_{\alpha\alpha}$ altered so that (2.5) is satisfied. The same is done for all $q_{\alpha\beta}$'s known to be 0. Denote the altered matrix by $\hat{\hat{Q}}$. Usually the elements of $\hat{Q}$ and $\hat{\hat{Q}}$ will differ only slightly. However, if the size of $\hat{\hat{Q}}$ is large and there are enough zeros in it, in the right places, then the subsequent computations involving powers of $\hat{\hat{Q}}$ will be considerably lightened.

A trial calculation is sufficient to show that $\hat{\hat{Q}}$ is not, in general the maximum likelihood estimate of Q, subject to the given restrictions. The estimate $\hat{\hat{Q}}$ has been chosen because it is the simplest reasonable estimate in sight. It can be shown that as $t' \to 0$ the relative asymptotic efficiency of the estimate $\hat{\hat{q}}_{ij}$ and the maximum likelihood estimate of q_{ij} converges to 1, for each element $\hat{\hat{q}}_{ij}$ of $\hat{\hat{Q}}$. Therefore, at least for t' small, the possible loss of efficiency in using $\hat{\hat{Q}}$ instead of the maximum likelihood estimate will be small.

On the other hand, $\hat{Q}$ and $\hat{\hat{Q}}$ are both consistent estimates of Q for case 2, so that the large sample distributions of the same rational functions (*e.g.*, $\hat{p}_{ij}(T)$ and $\hat{\hat{p}}_{ij}(T)$ defined below) of $\hat{Q}$ and $\hat{\hat{Q}}$ are the same. This follows from the theorems of sections 20.6 and 33.3 of Cramer's ('46) book. We are justified therefore in neglecting the distinction between $\hat{Q}$ and $\hat{\hat{Q}}$ when treating of the large sample distribution properties of such rational functions. We will, in fact, use the notation $\hat{Q}$ for either case 1 or case 2 estimates.

Starting with the estimate $\hat{Q}$ of Q, the estimate of $P(t)$ for any t, $0 \leq t \leq T$, is defined to be

$$\hat{P}(t) = I + \hat{Q}t + \hat{Q}^2 \frac{t^2}{2!} + Q^3 \frac{t^3}{3!} + \cdots \tag{4.8}$$

obtained by substituting $\hat{Q}$ into (2.3). Similarly substituting $\hat{Q}$ into (3.1) we obtain the estimate

$$\hat{e}(T_0) = IT_0 + \hat{Q}\frac{T_0{}^2}{2!} + Q^2 \frac{T_0{}^3}{3!} + \cdots \tag{4.9}$$

of $e(T_0)$. If some of the transition rates in $\hat{Q}$ are suppressed giving an estimated net rate matrix of the form $\hat{Q}_{\alpha\beta|\cdots|\mu\nu}$, then the corresponding net transition and net residence matrices are defined similarly. The suppression of rates in $\hat{Q}$ to obtain $\hat{Q}_{\alpha\beta|\cdots|\mu\nu}$ should not be confused with the suppression of rates in $\hat{Q}$ to obtain $\hat{\hat{Q}}$, the case 2 estimate. The latter is an estimate of Q, while $Q_{\alpha\beta|\cdots|\mu\nu}$ is not.

It will be observed, in forming the matrix products $(\hat{P}^*(t'))^n$ and $\hat{Q}^n$ for $n=1,2,\cdots$ that the sums of the elements of the rows of these powers equals 0. This provides a convenient check in computing.

5. THE LARGE SAMPLE DISTRIBUTION OF THE ESTIMATES.

The joint asymptotic distribution of the $\hat{q}_{ij}$'s will be found first. As explained in the previous section, it is sufficient to consider only the case 1 estimates. The only restrictions on these estimates are that $-\hat{q}_{ii}=\sum_{j\neq i}\hat{q}_{ij}$ for $i=1,\cdots,k$. Therefore, to avoid a singular distribution we consider the joint distribution only of the $\hat{q}_{ij}$'s for $i\neq j$. The distribution of the $\hat{q}_{ii}$'s can be found, if necessary, from the distribution of the $\hat{q}_{ij}$'s, $i\neq j$. The $\hat{q}_{ij}$'s for $i\neq j$ satisfy the regularity conditions of section 33. 3 of Cramer's ('46) book; hence we may conclude that these estimates

1. converge in probability to the corresponding elements of Q,

2. are jointly asymptotically normally distributed with the asymptotic covariance matrix equal to the inverse of the matrix

$$\left\{-\mathcal{E}\left(\frac{\partial^2 \log L}{\partial q_{\alpha\beta}\partial q_{\gamma\delta}}\right)\right\} \qquad \alpha,\beta,\gamma,\delta=1,\cdots,k;\ \alpha\neq\beta,\gamma\neq\delta$$

where $\mathcal{E}$ denotes "expected value of."

From (4. 5)

$$(5.1)\quad -\mathcal{E}\left(\frac{\partial^2 \log L}{\partial q_{\alpha\beta}^2}\right)=-\sum_{i=1}^{k}\sum_{j=1}^{k}\mathcal{E}(n_{ij})\left[-\frac{1}{p_{ij}(t')^2}\left(\frac{\partial p_{ij}(t')}{\partial q_{\alpha\beta}}\right)^2 + \frac{1}{p_{ij}(t')}\left(\frac{\partial^2 p_{ij}(t')}{\partial q_{\alpha\beta}^2}\right)\right].$$

To calculate $\mathcal{E}(n_{ij})$, first consider the path of a single patient, starting from time 0 in the initial state and classified subsequently at times $t', 2t',\cdots, st'=T$ the length of the study period. Let x_a be the number of transitions from state S_i at time $(a-1)t'$ to state S_j at time at'. Then the expected number of transitions $S_i\rightarrow S_j$ for the whole path is $\mathcal{E}(x_1+x_2+\cdots+x_s)=\mathcal{E}(x_1)+\cdots+\mathcal{E}(x_s)$. Now

$$\mathcal{E}(x_a)=p_{bi}((a-1)t')p_{ij}(t') \qquad a=1,\cdots,s$$

if S_b is the initial state. Therefore

$$\mathcal{E}(x_1+\cdots+x_s)=\sum_{a=1}^{s}p_{bi}((a-1)t')p_{ij}(t').$$

If there are n_b patients with the initial state S_b for $b=1,\cdots,k$ and n_{ij} is the total number of transitions $S_i\rightarrow S_j$ for all the $n=\sum n_b$ patients, then

$$\mathcal{E}(n_{ij})=\sum_{b=1}^{k}n_b\sum_{a=1}^{s}p_{bi}((a-1)t')p_{ij}(t').$$

Substituting in (5.1),

$$\begin{aligned}
-\mathcal{E}\left(\frac{\partial^2\log L}{\partial q_{\alpha\beta}{}^2}\right) &= -\sum_{i,j}\left\{p_{ij}(t')\left[\sum_b n_b\sum_{a=0}^{s-1}p_{bi}(at')\right]\right.\\
&\qquad \left.\cdot\left[\frac{-1}{p_{ij}{}^2(t')}\left(\frac{\partial p_{ij}(t')}{\partial q_{\alpha\beta}}\right)^2+\frac{1}{p_{ij}(t')}\left(\frac{\partial^2 p_{ij}(t')}{\partial q_{\alpha\beta}{}^2}\right)\right]\right\}\\
&=\sum_i\left[\sum_b n_b\sum_{a=0}^{s-1}p_{bi}(at')\right]\left[\sum_j\frac{1}{p_{ij}(t')}\left(\frac{\partial p_{ij}(t')}{\partial q_{\alpha\beta}}\right)^2\right]
\end{aligned}$$

$$=\sum_b n_b\left\{\sum_i\left[\sum_{a=0}^{s-1}p_{bi}(at')\right]\left[\sum_j\frac{1}{p_{ij}(t')}\left(\frac{\partial p_{ij}(t')}{\partial q_{\alpha\beta}}\right)^2\right]\right\}. \tag{5.2}$$

If t' is sufficiently small while T remains fixed, this last expression can be approximated by one of simpler form, for we have

$$\lim_{\substack{t'\to 0\\ st'=T}} t'\sum_{a=0}^{s-1}p_{bi}(at')=e_{bi}(T)$$

$$\lim_{\substack{t'\to 0\\ st'=T}}\sum_j\frac{1}{t'p_{ij}(t')}\left(\frac{\partial p_{ij}(t')}{\partial q_{\alpha\beta}}\right)^2=\begin{cases}\dfrac{1}{q_{\alpha\beta}} & \text{if } i=\alpha\\[2mm] 0 & i\neq\alpha\end{cases}$$

using l'Hospital's rule for the second limit. Therefore

$$\lim_{\substack{t'\to 0\\ st'=T}} -\mathcal{E}\left(\frac{\partial^2\log L}{\partial q_{\alpha\beta}{}^2}\right)=\frac{1}{q_{\alpha\beta}}\sum_b n_b e_{b\alpha}(T). \tag{5.3}$$

The expressions (5.2) and (5.3) have been calculated for two systems of transition rates and for several values of t' in order to give some idea of the effect of t' on the accuracy of the approximation (5.3). The first system is one used as an example by Fix and Neyman. This system has four states with the rate matrix

$$Q = \begin{bmatrix} -3.7 & 1.0 & 2.7 & 0 \\ 0 & 0 & 0 & 0 \\ .2 & 0 & -.4 & .2 \\ 0 & 0 & 0 & 0 \end{bmatrix}.$$

The interpretation of the states is of no consequence for this illustration. For this system it is assumed that all patients have the initial state S_1.

The second system was chosen arbitrarily and consists of three states with the rate matrix

$$Q = \begin{bmatrix} -.65 & .45 & .20 \\ 1.20 & -1.97 & .77 \\ .33 & .68 & -1.01 \end{bmatrix}.$$

It is assumed that the initial states are divided equally among the three states.

TABLE 1

Asymptotic Variances of Estimates of Transition Rates for Two Systems of True Values of Rates

TRUE RATES		PROPORTION t' OF TOTAL PERIOD OF OBSERVATION T BETWEEN SUCCESSIVE CLASSIFICATIONS			
		$t' = \frac{1}{6}T$	$t' = \frac{1}{10}T$	$t' = \frac{1}{20}T$	$t' \to 0$
System* I	$q_{12} = 1.0$	$3.59/n$	$3.57/n$	$3.56/n$	$3.56/n$
	$q_{13} = 2.7$	$10.30/n$	$9.92/n$	$9.74/n$	$9.60/n$
	$q_{31} = .2$	$.60/n$	$.52/n$	$.47/n$	$.42/n$
	$q_{34} = .2$	$.42/n$	$.42/n$	$.42/n$	$.42/n$
System** II	$q_{12} = .45$		$.42/n$	$.38/n$	$.35/n$
	$q_{13} = .20$		$.19/n$	$.17/n$	$.16/n$
	$q_{21} = 1.20$		$1.72/n$	$1.63/n$	$1.54/n$
	$q_{23} = .77$		$1.13/n$	$1.06/n$	$.99/n$
	$q_{31} = .33$		$.42/n$	$.38/n$	$.35/n$
	$q_{32} = .68$		$.83/n$	$.77/n$	$.72/n$

* For system I, n is the number of patients with initial state S_1, the only initial state.

** For system II, equal numbers n of patients have the initial states S_1, S_2, and S_3.

We find, similarly, for the asymptotic covariances

$$(5.4)\qquad -\mathcal{E}\left(\frac{\partial^2 \log L}{\partial q_{\alpha\beta}\partial q_{\gamma\delta}}\right) = \sum_b n_b \left\{ \sum_i \left[\sum_{a=0}^{s-1} p_{bi}(at') \right] \right.$$

$$\left. \cdot \left[\sum_j \frac{1}{p_{ij}(t')} \left(\frac{\partial p_{ij}(t')}{\partial q_{\alpha\beta}}\right)\left(\frac{\partial p_{ij}(t')}{\partial q_{\gamma\delta}}\right)\right] \right\}$$

$$= 0 \text{ as } t' \to 0,\ s \to \infty, \text{ and } st' = T.$$

We conclude that for n large and t' small the $\hat{q}_{\alpha\beta}$'s for $\alpha \neq \beta$ are approximately normally independently distributed with the mean of $\hat{q}_{\alpha\beta}$ equal to $q_{\alpha\beta}$ and the variance equal to

$$(5.5)\qquad \frac{q_{\alpha\beta}}{\sum_b n_b e_{b\alpha}(T)}$$

in this normal distribution.

Estimates of (5. 2), (5. 4) and (5. 5) are obtained by substituting sample estimates for the corresponding parameters. For example, in (5. 2), $\hat{p}_{bi}(at')$, $\hat{p}_{ij}(t')$ and $\frac{\partial \hat{p}_{ij}(t')}{\partial \hat{q}_{\alpha\beta}}$ are substituted for $p_{bi}(at')$, $p_{ij}(t')$, and $\frac{\partial p_{ij}(t')}{\partial q_{\alpha\beta}}$ respectively.

Turning now to $\hat{p}_{ij}(t)$ and $\hat{e}_{ij}(t)$ for $0 \leq t \leq T$, it can be shown that these estimates are asymptotically normally distributed. The asymptotic mean of $\hat{p}_{ij}(t)$ is $p_{ij}(t)$ and the asymptotic variance is

$$(5.6)\qquad \frac{1}{n} \sum_{\alpha\neq\beta} \sum_{\gamma\neq\delta} \left(\frac{\partial p_{ij}(t)}{\partial q_{\alpha\beta}}\right)\left(\frac{\partial p_{ij}(t)}{\partial q_{\gamma\delta}}\right) c'_{\alpha\beta\gamma\delta}(n),$$

where the derivatives are evaluated at the true values of $q_{\alpha\beta}$, $q_{\gamma\delta}$, etc., and $\frac{1}{n}\, c'_{\alpha\beta\gamma\delta}(n)$ is the asymptotic covariance of $\hat{q}_{\alpha\beta}$ and $\hat{q}_{\gamma\delta}$. For t' (the interval between successive observations) small this is approximately

$$(5.7)\qquad \sum_{\alpha\neq\beta} \left(\frac{\partial p_{ij}(t)}{\partial q_{\alpha\beta}}\right)^2 \frac{q_{\alpha\beta}}{\sum_b n_b e_{b\alpha}(T)}.$$

The asymptotic mean of $\hat{e}_{ij}(t)$ is $e_{ij}(t)$, while the exact and approximate asymptotic variances, corresponding to (5. 6) and (5. 7), are

$$(5.8)\qquad \frac{1}{n} \sum_{\alpha\neq\beta} \sum_{\gamma\neq\delta} \left(\frac{\partial e_{ij}(t)}{\partial q_{\alpha\beta}}\right)\left(\frac{\partial e_{ij}(t)}{\partial q_{\gamma\delta}}\right) c'_{\alpha\beta\gamma\delta}(n)$$

and

$$\sum_{\alpha \neq \beta} \left(\frac{\partial e_{ij}(t)}{\partial q_{\alpha\beta}} \right)^2 \frac{q_{\alpha\beta}}{\sum_b n_b e_{b\alpha}(T)} . \tag{5.9}$$

The proof is deferred to appendix 3. Estimates of (5.6), (5.7), (5.8), and (5.9) are found by substituting $\hat{q}_{\alpha\beta}$ for $q_{\alpha\beta}$ throughout.

In computing the derivatives in the preceding formulas, the matrices

$$\left\{ \frac{\partial p_{ij}(t)}{\partial q_{\alpha\beta}} \right\}, \left\{ \frac{\partial e_{ij}(t)}{\partial q_{\alpha\beta}} \right\}$$

can be computed as follows: For any matrix $A = \{a_{ij}(x)\}$ define the derivative of A with respect to x as the matrix of the derivatives of its elements, *i.e.*,

$$\frac{\partial A}{\partial x} = \left\{ \frac{\partial a_{ij}(x)}{\partial x} \right\} .$$

We have then,

$$\frac{\partial P(t)}{\partial q_{\alpha\beta}} = \frac{\partial Q}{\partial q_{\alpha\beta}} t + \left(\frac{\partial Q}{\partial q_{\alpha\beta}} Q + Q \frac{\partial Q}{\partial q_{\alpha\beta}} \right) \frac{t^2}{2!} + \left(\frac{\partial Q^2}{\partial q_{\alpha\beta}} Q + Q^2 \frac{\partial Q}{\partial q_{\alpha\beta}} \right) \frac{t^3}{3!} + \cdots . \tag{5.10}$$

Letting

$$\begin{aligned} A_1 &= \frac{\partial Qt}{\partial q_{\alpha\beta}} \\ A_2 &= \tfrac{1}{2} A_1 Qt + \tfrac{1}{2} Qt A_1 \\ A_3 &= \tfrac{1}{3} A_2 Qt + \tfrac{1}{3} \left(\frac{Q^2 t^2}{2!} \right) A_1 \\ A_4 &= \tfrac{1}{4} A_3 Qt + \tfrac{1}{4} \left(\frac{Q^3 t^3}{3!} \right) A_1 \\ &\cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot \end{aligned}$$

(5.10) becomes

$$\frac{\partial P(t)}{\partial q_{\alpha\beta}} = A_1 + A_2 + A_3 + \cdots . \tag{5.11}$$

We find similarly

$$\frac{\partial e(t)}{\partial q_{\alpha\beta}} = A_1 \frac{t}{2} + A_2 \frac{t}{3} + A_3 \frac{t}{4} + \cdots . \tag{5.12}$$

6. TESTS OF HYPOTHESES

In this section we are concerned with two processes, having the same sets of states but with possibly different rate (and hence different transition and residence) matrices. We shall distinguish between the two processes, in our notation, by means of the superscripts "(1)", and "(2)".

Some questions that might arise in connection with a follow-up study are:

1. Are the rate matrices of two periods the same or not? Such a question might arise in deciding whether to combine two adjacent periods into a single one of length equal to the sum of the two original periods. Or it might arise in deciding whether to combine two sets of data, possibly collected at different places, representing parallel studies.

2. If $p^{(1)}(T)$ and $p^{(2)}(T)$ are transition probabilities for the period $[0, T]$, corresponding to two different treatments (and hence two different processes), are they the same?

3. The same question as the previous one with respect to $e^{(1)}(T)$ and $e^{(2)}(T)$.

Considering question one first, an approximate large sample test of the null hypothesis that the rates $q_{\alpha\beta}{}^{(1)}$ and $q_{\alpha\beta}{}^{(2)}$ with $\alpha \neq \beta$ are equal, against the alternative hypothesis that $q_{\alpha\beta}{}^{(1)} \neq q_{\alpha\beta}{}^{(2)}$, is based on the statistic

$$U_{\alpha\beta} = \frac{|\hat{q}_{\alpha\beta}{}^{(1)} - \hat{q}_{\alpha\beta}{}^{(2)}|}{\sqrt{\sigma^2(\hat{q}_{\alpha\beta}{}^{(1)}) + \sigma^2(\hat{q}_{\alpha\beta}{}^{(2)})}} \tag{6.1}$$

where $\sigma^2(\hat{q}_{\alpha\beta}{}^{(1)})$ and $\sigma^2(\hat{q}_{\alpha\beta}{}^{(2)})$ are (estimates of) the asymptotic variances of $\hat{q}_{\alpha\beta}{}^{(1)}$ and $\hat{q}_{\alpha\beta}{}^{(2)}$ respectively. These are given by (5.2), or (5.3) if t' is small. From section 5 it follows that $U_{\alpha\beta}$ is distributed approximately as the absolute value of a unit normal variate. The hypothesis of the equality of $q_{\alpha\beta}{}^{(1)}$ and $q_{\alpha\beta}{}^{(2)}$ is rejected for a large value of $U_{\alpha\beta}$ (depending on the significance level). It will be noted that $T^{(1)}$ need not equal $T^{(2)}$.

For t' small we can also test the equality of several rates jointly. Let the null hypothesis be that

$$q_{\alpha_1\beta_1}{}^{(1)} = q_{\alpha_1\beta_1}{}^{(2)}, q_{\alpha_2\beta_2}{}^{(1)} = q_{\alpha_2\beta_2}{}^{(2)}, \cdots, q_{\alpha_p\beta_p}{}^{(1)} = q_{\alpha_p\beta_p}{}^{(2)},$$

where $\alpha_i \neq \beta_i$ for $i = 1, \cdots, p$ and the (α_i, β_i)'s are distinct, and the alternative hypothesis be that at least one of the equalities fails to hold.

Since the $\hat{q}_{\alpha\beta}$'s are asymptotically independent as $n \to \infty$ and $t' \to 0$, it follows that for large n and small t'

$$\sum_{i=1}^{p} U_{\alpha_i\beta_i}^{2} = \chi_p^{2} \tag{6.2}$$

is distributed approximately as χ^2 with p degrees of freedom. In particular, we can test the hypothesis that $Q^{(1)} = Q^{(2)}$ against the alternative that $Q^{(1)} \neq Q^{(2)}$ by summing (6.2) over all (α, β)'s for which $\alpha \neq \beta$. The hypothesis of equality is rejected for a large value of χ_p^2 (depending on the significance level).

The significance levels of the tests based on (6.1) and (6.2) should not be taken too literally, as the tests are based on several assumptions: the normal distributions assumed for the $\hat{q}_{\alpha\beta}$'s are asymptotic, not exact; the variances in the denominator of (6.1) are estimates, not exact expressions; and unless $t' = 0$, the $\hat{q}_{\alpha\beta}$'s are only approximately independent, asymptotically. Nothing much more specific can be said about the effect on the significance level without inquiring more deeply into the distributions of the statistics (6.1) and (6.2), or conducting some sampling experiments.

With respect to the elements of the transition and residence matrices for $T^{(1)} = T^{(2)} = T$, statistics similar to (6.1) can be used for large sample tests of hypotheses of the form $p_{\alpha\beta}^{(1)}(T) = p_{\alpha\beta}^{(2)}(T)$ or $e_{\alpha\beta}^{(1)}(T) = e_{\alpha\beta}^{(2)}(T)$ for $\alpha \neq \beta$, against either one or two sided alternatives of inequality. For example, to test the hypothesis that $p_{\alpha\beta}^{(1)}(T) = p_{\alpha\beta}^{(2)}(T)$ against the alternative that $p_{\alpha\beta}^{(1)}(T) > p_{\alpha\beta}^{(2)}(T)$ we may use the statistic

$$V_{\alpha\beta} = \frac{\hat{p}_{\alpha\beta}^{(1)}(T) - \hat{p}_{\alpha\beta}^{(2)}(T)}{\sqrt{\sigma^2(\hat{p}_{\alpha\beta}^{(1)}(T)) + \sigma^2(\hat{p}_{\alpha\beta}^{(2)}(T))}}, \tag{6.3}$$

which is approximately normally distributed with zero mean and unit variance, for large n. The hypothesis of equality is rejected for large positive values of $V_{\alpha\beta}$. Here again $\sigma^2(\hat{p}_{\alpha\beta}^{(1)}(T))$ and $\sigma^2(\hat{p}_{\alpha\beta}^{(2)}(T))$ are (estimates of) the asymptotic variances of $\hat{p}_{\alpha\beta}^{(1)}(T)$ and $\hat{p}_{\alpha\beta}^{(2)}(T)$ respectively, and these variances are given by expressions of the form (5.6), or (5.7) if t' is small.

7. TESTS OF GOODNESS OF FIT OF THE MODEL

We have so far dealt with tests of hypotheses assuming that the model fits the disease process adequately. There are certain internal checks (*i.e.*, checks based on the data alone) of the goodness of fit of the model that can be made. These checks are of conditions 2 and 3 of section 2.

Condition 2 is: the probability that a patient in state S_i at any time during the study, is subsequently in state S_j at a later time in the study, depends on the two states in question but not on the previous history of the patient. As an alternative to condition 2, we may take the hypothesis: if a patient at times t_{-1} and t_0 $(t_{-1} < t_0)$ is in states S_h and S_i, respectively, then the probability of his being in state S_j at time t_1 $(t_0 < t_1; t_{-1}, t_0, t_1$ any times in the period of observation) depends on S_h as well as on S_i and S_j, for at least one (triple S_h, S_i, S_j) of states.

A test of condition 2 against this alternative is the following. Assuming that the interval between successive classifications of n patients is t', let n_{hij} be the number of transitions $S_h \rightarrow S_i \rightarrow S_j$. Therefore $\sum_h n_{hij} = n_{ij}$. If condition 2 is satisfied, then for each pair (i, j),

$$\hat{p}_{hij}(t') = \frac{n_{hij}}{\sum_{j=1}^{k} n_{hij}} \qquad h = 1, \cdots, k, \tag{7.1}$$

have the same expected values; if the alternative hypothesis is true then for at least one pair (i, j), the expected values of $\hat{p}_{hij}(t')$ for $h = 1, \cdots, k$ will not be the same. Accordingly, we may use a χ^2 test of homogeneity of proportions. It will be observed that if, and only if, q_{ii} is zero, then the proportions (7.1) are either identically one or zero for all samples. Letting E be the set of indices i for which q_{ii} is not *a priori* zero, we have

$$\chi_u^2 = \sum_{h \varepsilon E} \sum_{i \varepsilon E} \sum_{j=1}^{k} \frac{(n_{hij} - \mathcal{E} n_{hij})^2}{\mathcal{E} n_{hij}} \tag{7.2}$$

$$\mathcal{E} n_{hij} = \frac{\left(\sum_{j=1}^{k} n_{hij}\right)\left(\sum_{h \varepsilon E} n_{hij}\right)}{\sum_{h \varepsilon E} \sum_{j=1}^{k} n_{hij}}.$$

If E has m elements, then n, the degrees of freedom, equals $m[(k-1)m-(k-1)]$. A large value of χ_u^2 renders condition 2 suspect.

The distribution of χ_u^2 is asymptotically χ^2 as $n \rightarrow \infty$. For finite n the approximation is poor if any of the n_{hij}'s or $(\sum_j n_{hij})$'s are small. If any of these quantities are small, it will be better to partition the n_{ij}'s into fewer than m parts. Suppose we partition the set E into

$b < m$ subsets, $E_1, E_2, \cdots, E_b$. Define $n_{E_h ij}$ as the number of transitions $S_\alpha \rightarrow S_i \rightarrow S_j$, where $\alpha \varepsilon E_h$. The formulae of (7.2) are then replaced by

$$\chi_v^2 = \sum_{h=1}^{b} \sum_{i \varepsilon E} \sum_{j=1}^{k} \frac{(n_{E_h ij} - \mathcal{E} n_{E_h ij})^2}{\mathcal{E} n_{E_h ij}} \tag{7.3}$$

$$\mathcal{E} n_{E_h ij} = \frac{(\sum_{j=1}^{k} n_{E_h ij})(\sum_{h=1}^{b} n_{E_h ij})}{\sum_{h=1}^{b} \sum_{j=1}^{k} n_{E_h ij}}$$

while $v = m[(k-1)b - (k-1)]$.

However, it may happen that no matter how the n_{ij}'s are partitioned there will be a sum $\sum_{h=1} n_{E_h ij}$ or an expectation $\mathcal{E} n_{E_h ij}$ that is too small. In this case, an alternative test can sometimes be used. The indices $1, \cdots, k$ are partitioned into d subsets $(2 \leq d \leq k), F_1, F_2, \cdots, F_d$ and $n_{F_h ij}$ defined to be the number of transitions $S_\alpha \rightarrow S_i \rightarrow S_j$ where $\alpha \varepsilon F_h$. The matrix $\{n_{ij}\}$ is thus partitioned into d matrices $\{n_{F_h ij}\}$, $(h = 1, \cdots, d)$, from which are calculated matrices Q_h, $(h = 1, \cdots, d)$. The Q_h's are then tested for equality pairwise by the test of the previous section. This check involves considerable computation for $d > 2$, however.

If condition 3 is violated, one way in which it is likely to be violated is that some of the elements of Q undergo an increasing or decreasing trend with time. The ordinary life table regarded as a two state process with the states "alive" and "dead" is an example of a process which violates condition 3 in this manner, if we take as the total period of observation the first five years. Here the transition probability from the state "alive" to the state "dead" for one year decreases considerably from the first to the last year. The condition is better met for five years in the middle of the table. As a test of condition 3 against the alternative of a trend, the total period of the study can be divided in half and $n_{ij}^{(1)}$ and $n_{ij}^{(2)}$ computed for the first and second halves respectively for $i \varepsilon E$ and $j = 1, \cdots, k$, where E is defined above. Again, we use a χ^2 test of homogeneity with

$$\chi_w^2 = \sum_{h=1}^{2} \sum_{i \varepsilon E} \sum_{j=1}^{k} \frac{(n_{ij}^{(h)} - \mathcal{E} n_{ij}^{(h)})^2}{\mathcal{E} n_{ij}^{(h)}} \tag{7.6}$$

$$\mathcal{E} n_{ij}^{(h)} = \frac{(\sum_{j=1}^{k} n_{ij}^{(h)})(\sum_{h=1}^{2} n_{ij}^{(h)})}{\sum_{j=1}^{k} \sum_{h=1}^{2} n_{ij}^{(h)}}.$$

Here $w = m[(k-1)2-(k-1)]$. Alternatively, we can compute $\hat{Q}^{(1)}$ and $\hat{Q}^{(2)}$ for the first and second halves respectively and test these matrices for equality.

What do we do if these tests show that condition 2 or 3 is apparently violated?

If condition 3 is violated, the total period can be divided into sub-periods small enough for the rate matrices of each sub-period to satisfy condition 3 approximately. The method of section 8 is then used to join the sub-periods together.

If condition 2 is violated, it may be possible to substitute a higher order Markov process, *i. e.*, one in which the transition probabilities are determined by two or more preceding states. The generalization of the method of this paper to such a higher order model is straightforward, but will not be done here.

8. TREATMENT OF TWO CONTIGUOUS STUDY PERIODS

Assume that there are k states for each period and that the first period is from $t=0$ to $t=T_1$ and the second from $t=T_1$ to $t=T_2$. For the first period, let Q_1 be the rate matrix,

$$P_1(t) = \{p_{ij}^{(1)}(t)\} \qquad 0 \leq t \leq T_1$$

the corresponding transition matrix, and

$$e_1(t') = \{e_{ij}^{(1)}(t')\} = \int_0^{t'} P_1(t)\,dt \qquad 0 \leq t' \leq T_1 \tag{8.1}$$

the residence matrix. For the second period, let the corresponding matrices be Q_2,

$$P_2(t-T_1) = \{p_{ij}^{(2)}(t+T_1)\} \qquad T_1 \leq t \leq T_2$$

and

$$e_2(t'-T) = \{e_{ij}^{(2)}(t'-T_1)\} = \int_{T_1}^{t'} P_2(t-T_1)\,dt \qquad T_1 \leq t' \leq T_2. \tag{8.2}$$

It follows that if at time 0 an individual is in state S_i, the probability $p_{ij}(t)$ of being in state S_j at time t is

$$p_{ij}(t) = \begin{cases} p_{ij}^{(1)}(t) & 0 \leq t \leq T_1 \\ \sum_{\alpha=1}^{k} p_{i\alpha}^{(1)}(T_1)\,p_{\alpha j}^{(2)}(t-T_1) & T_1 \leq t \leq T_2. \end{cases}$$

In matrix notation, this is

$$(8.3) \qquad P(t)=\begin{cases} P_1(T) & 0\leq t\leq T_1 \\ P_1(T_1)P_2(t-T_1) & T_1\leq t\leq T_2. \end{cases}$$

In terms of Q_1 and Q_2,

$$(8.4) \quad P(t)=\begin{cases} I+Q_1t+Q_1^2\frac{t^2}{2!}+\cdots & 0\leq t\leq T_1 \\ I+[Q_1T_1+Q_2(t-T_1)]+[Q_1T_1+Q_2(t-T_1)]^2\frac{1}{2!} & \\ \quad +[Q_1T_1+Q_2(t-T_1)]^3\frac{1}{3!}+\cdots & T_1\leq t\leq T_2. \end{cases}$$

From (8.1), (8.2) and (8.3), we have, for $T_1\leq t'\leq T_2$,

$$\begin{aligned}(8.5) \qquad e(t')&=\int_0^{t'}P(t)\,dt=\int_0^{T_1}P(t)\,dt+\int_{T_1}^{t'}P(t)\,dt \\ &=\int_0^{T_1}P_1(t)\,dt+P_1(t)\int_{T_1}^{t'}P_2(t-T_1)\,dt \\ &=e_1(T_1)+P_1(T_1)e_2(t'-T_1).\end{aligned}$$

As in the case of a single period of study, these formulae hold also for the estimates $\hat{Q}_1, \hat{Q}_2, \hat{P}_1(t),\cdots$ or net estimates

$$\hat{Q}_{1\cdot\alpha\beta|\gamma\delta|\ldots}, \hat{Q}_{2\cdot\alpha\beta|\gamma\delta|\ldots}, \hat{P}_{1\cdot\alpha\beta|\gamma\delta|\ldots}(t), \text{etc.}$$

The results of section 4 concerning the large sample distribution of the $\hat{p}_{ij}(t)$'s and $\hat{e}_{ij}(t)$'s extend without any difficulty to the present case, and we may state that for any pair of indices $i, j=1,\cdots,k$, $\hat{p}_{ij}(t)$, $T_1\leq t\leq T_2$ is asymptotically normally distributed with asymptotic mean $p_{ij}(t)$ and asymptotic variance

$$\begin{aligned}(8.6) \qquad &\frac{1}{n^{(1)}}\sum_{\alpha\neq\beta}\sum_{\gamma\neq\delta}\left(\frac{\partial p_{ij}(t)}{\partial q_{\alpha\beta}^{(1)}}\right)\left(\frac{\partial p_{ij}(t)}{\partial q_{\gamma\delta}^{(1)}}\right)c'_{\alpha\beta\gamma\delta}{}^{(1)}(n^{(1)}) \\ &\quad +\frac{1}{n^{(2)}}\sum_{\alpha\neq\beta}\sum_{\gamma\neq\delta}\left(\frac{\partial p_{ij}(t)}{\partial q_{\alpha\beta}^{(2)}}\right)\left(\frac{\partial p_{ij}(t)}{\partial q_{\gamma\delta}^{(2)}}\right)c'_{\alpha\beta\gamma\delta}{}^{(2)}(n^{(2)}).\end{aligned}$$

For t' small we may use approximations of the form (5.7). The formula (8.6) is the generalization of (5.6).

The matrix

$$\frac{\partial P(t)}{\partial q_{\alpha\beta}^{(h)}} \qquad T_1\leq t\leq T_2,\ h=1,2$$

can be found from (5.11) using the relations

$$\frac{\partial P(t)}{\partial q_{\alpha\beta}^{(1)}} = \frac{\partial P_1(T_1)}{\partial q_{\alpha\beta}^{(1)}} P_2(t-T_1)$$

$$\frac{\partial P(t)}{\partial q_{\gamma\delta}^{(2)}} = P_1(T_1)\frac{\partial P_2(t-T_1)}{\partial q_{\gamma\delta}^{(2)}}$$

derived from (8.3).

Similarly $\hat{e}_{ij}(t)$ for $T_1 \leq t \leq T_2$ and $i, j = 1, \cdots, k$ is asymptotically normally distributed with asymptotic mean $e_{ij}(t)$ and with the asymptotic variance given by an expression of the same form as (8.6) but with $e_{ij}(t)$ replacing $p_{ij}(t)$. The same remark as before applies if t' is small. Furthermore

$$\frac{\partial e(t)}{\partial q_{\alpha\beta}^{(h)}} \qquad T_1 \leq t \leq T_2,\ h = 1, 2$$

is computed using (5.11) and (5.12) and the relations

$$\frac{\partial e(t)}{\partial q_{\alpha\beta}^{(1)}} = \frac{\partial e_1(t)}{\partial q_{\alpha\beta}^{(1)}} + \frac{\partial P_1(T_1)}{\partial q_{\alpha\beta}^{(1)}} e_2(t-T_1)$$

$$\frac{\partial e(t)}{\partial q_{\gamma\delta}^{(2)}} = P_1(T_1)\frac{\partial e_2(t-T_1)}{\partial q_{\gamma\delta}^{(2)}}$$

derived from (8.5).

An estimate of (8.6) is obtained in the usual way by substituting estimates of the p's and q's for these parameters throughout.

9. EXAMPLE AND COMPARISON OF FIX AND NEYMAN'S AND OUR ESTIMATES

We consider in this section a single period only. It was not possible to obtain any actual data with which to illustrate the estimates of this paper. Instead, samples were generated artificially based on a system of parameters (already given in section 5) used by Fix and Neyman ('51). To generate the samples, the Fisher and Yates ('49) tables of random digits, and the Rand Corporation series of random digits appearing in recent issues of the *Journal of the American Statistical Association* were used.

The samples so obtained enable us to compare the estimates of Fix and Neyman with ours to give an indication of the relative performances of the estimates. As a basis for comparison, the sample variances of the estimates of the risks, for the two estimates in question, have been used. As the estimates of this paper are based on somewhat more information than Fix and Neyman's estimates, it is to be expected that the sample variances of our estimates will tend to be less. The question of greater interest is how much less; unfortunately the present experiment is too small to answer this question.

The example that we use has the rate matrix

$$\begin{bmatrix} -3.7 & 1.0 & 2.7 & 0 \\ 0 & 0 & 0 & 0 \\ .2 & 0 & -.4 & .2 \\ 0 & 0 & 0 & 0 \end{bmatrix}.$$

Briefly, the interpretation is:

S_1: active cancer
S_2: death from cancer or operative death
S_3: cured or apparently well
S_4: lost from observation or death from other causes.

The *a priori* assumptions are that $q_{14}=q_{22}=q_{32}=q_{44}=0$. Letting the total period of observation equal 1, and observing the "patients" at intervals of 1/10, the true transition matrix for the interval 1/10 is, by (2.3),

$$P(1/10)=\begin{bmatrix} .693 & .084 & .221 & .002 \\ 0 & 1 & 0 & 0 \\ .016 & .001 & .963 & .020 \\ 0 & 0 & 0 & 1 \end{bmatrix}.$$

On the basis of $P(1/10)$, a total of 3000 paths were generated, half with the initial state S_1 and half with the initial state S_3. For example, the first four of these paths are:

$$\begin{matrix} 1 & 1 & 1 & 3 & 3 & 3 & 3 & 3 & 3 & 3 \\ 1 & 1 & 3 & 3 & 3 & 4 & 4 & 4 & 4 & 4 \\ 1 & 1 & 3 & 3 & 3 & 3 & 3 & 3 & 3 & 3 \\ 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1. \end{matrix}$$

The 3000 paths were divided up into 20 groups. The first ten groups each contained 50 paths with initial state S_1 and 50 with initial state S_3; the second 10 groups each contained 100 paths with initial state S_1 and 100 with initial state S_3. The paths were subdivided into these groups according to their order of generation. Following the notation of section 4, table 2 gives the numbers of transitions $S_i \to S_j$ for each sample. It will be observed that the remaining n_{ij}'s are *a priori* 0 or 1.

The *a priori* restrictions on Q are such that q_{12}, q_{13}, q_{31}, and q_{34} determine Q, and therefore it is sufficient to give the estimates of these parameters only. Our estimates of these parameters are based on (4.7), while Fix and Neyman's estimates are based on the formulae of page 230 of their paper. It will be noted that their state S_i is our state S_{i+1} for $i = 0, 1, 2, 3$. The estimates for the 20 samples are given in table 3.

TABLE 2

Numbers of Transitions in Twenty Samples

SAMPLE	n_{11}	n_{12}	n_{13}	n_{14}	n_{31}	n_{32}	n_{33}	n_{34}
1	124	16	37	0	10	0	638	13
2	165	13	40	0	8	1	636	11
3	142	15	40	0	8	1	611	15
4	137	22	29	0	5	1	597	11
5	140	13	36	0	6	0	620	16
6	126	12	40	1	6	1	666	15
7	114	12	37	0	6	0	642	16
8	153	13	43	0	12	0	609	15
9	110	16	36	1	4	0	619	15
10	140	16	37	0	10	2	609	17
11	238	44	66	2	19	1	1170	33
12	236	38	79	0	23	0	1246	24
13	231	24	89	1	23	2	1339	20
14	235	31	78	1	16	0	1273	29
15	271	35	81	0	26	1	1241	24
16	288	25	83	2	24	1	1300	22
17	232	20	85	0	13	1	1303	35
18	239	34	74	2	19	1	1286	22
19	293	28	80	1	20	0	1286	24
20	255	26	81	1	15	2	1264	30

Fix and Neyman's estimates for samples 6 and 12 are infinite and have therefore been omitted.

In table 4 are given the sample variances of the transition rates based on the two groups of samples, 1 to 10, and 11 to 20. It will be seen from table 4 that the estimates of this paper, with one exception, have the smaller variances.

In practice, one would also be interested in the matrices $P(1)$ and $e(1)$, and therefore in the variances of the estimates of these two matrices. It can be shown, however, that the variances of the $\hat{p}_{ij}(1)$'s and $\hat{e}_{ij}(1)$'s are approximately equal to linear combinations, with positive coefficients, of the variances of the $\hat{q}_{ij}$'s, so that for an indication of the performance of the estimates in general, it is sufficient to examine the sample variances of the $\hat{q}_{ij}$'s.

TABLE 3

Estimates of Transition Rates: (A) *Ours,* (B) *Fix and Neyman's*

	ESTIMATES OF							
	$q_{12} = 1.0$		$q_{13} = 2.7$		$q_{31} = .2$		$q_{34} = .2$	
SAMPLE*	A	B	A	B	A	B	A	B
1	1.08	1.06	2.54	2.21	.18	.17	.20	.13
2	.68	.79	2.14	2.04	.14	.00	.17	.22
3	.89	1.20	2.43	2.71	.15	.05	.24	.38
4	1.36	1.34	1.83	1.88	.10	.08	.18	.14
5	.80	.67	2.25	1.64	.11	.06	.25	.16
6	.79	—	2.70	—	.11	—	.22	—
7	.88	.72	2.75	2.10	.11	.15	.26	.13
8	.73	.96	2.45	2.95	.23	.47	.24	.04
9	1.19	1.28	2.72	2.63	.08	.00	.24	.36
10	.97	.94	2.30	2.28	.19	.35	.27	.01
11	1.52	1.37	2.33	2.13	.19	.21	.28	.29
12	1.31	—	2.78	—	.22	—	.19	—
13	.84	1.02	3.19	3.58	.21	.12	.15	.20
14	1.08	1.12	2.78	2.79	.15	.21	.22	.16
15	1.08	1.40	2.55	3.21	.24	.25	.19	.13
16	.74	.84	2.49	3.07	.21	.44	.17	—.02
17	.71	.60	3.09	2.62	.12	.04	.26	.33
18	1.17	1.05	2.59	2.49	.18	.18	.17	.11
19	.81	.84	2.37	2.38	.18	.13	.18	.23
20	.85	1.00	2.70	2.91	.14	.10	.23	.19

* Samples 1-10 consist of 100 paths, half with initial state S_1 and half with initial state S_3. Samples 11-20 have twice as many paths in each category.

Besides $P(1)$ and $e(1)$ the other matrices of interest in this example are $P_{34}(1)$ and $e_{34}(1)$, which are the transition and residence matrices with the "inessential" risk q_{34} suppressed. The estimates of $P(1)$, $e(1)$, $P_{34}(1)$, and $e_{34}(1)$ have been computed from (2.3) and (3.1), only for samples 1 and 10, and only for the estimates of this paper. These estimates are given in table 5. Together with estimates of their variances obtained from (5.7) and (5.9), they are examples of solutions of the estimation problem in our model.

TABLE 4

Sample Variances about True Values, of Estimates of Transition Rates, Based on Two Sample Sizes of (A) *Our Estimates and* (B) *Fix and Neyman's Estimates*

SAMPLE VARIANCE OF	ESTIMATE	SAMPLE SIZE = TOTAL NUMBER OF PATHS IN EACH SAMPLE		NUMBER OF SAMPLES
		100	200	
$\hat{q}_{12}$	A	.046	.064	10
	B	.052	.058	9
$\hat{q}_{13}$	A	.160	.073	10
	B	.340	.192	9
$\hat{q}_{31}$	A	.006	.002	10
	B	.026	.012	9
$\hat{q}_{34}$	A	.002	.002	10
	B	.015	.010	9

TABLE 5

True Values and Our Estimates of $P(1)$, $e(1)$, $P_{34}(1)$, *and* $e_{34}(1)$

ESTIMATE OF	SAMPLE 1	SAMPLE 10	WITH RATE q_{34} SUPPRESSED ESTIMATE OF *	SAMPLE 1	SAMPLE 10
$p_{11}(1) = .054$	.053	.043			
$p_{12}(1) = .281$	.307	.406	$p_{11.34}(1) = .036$	.057	.047
$p_{13}(1) = .570$	.549	.444	$p_{12.34}(1) = .324$	.308	.408
$p_{14}(1) = .095$	.091	.106	$p_{13.34}(1) = .641$	.636	.545
$p_{31}(1) = .042$	.040	.036			
$p_{32}(1) = .035$	.035	.048	$p_{31.34}(1) = .047$	.046	.045
$p_{33}(1) = .751$	.752	.687	$p_{32.34}(1) = .086$	.038	.054
$p_{34}(1) = .172$	.173	.228	$p_{33.34}(1) = .866$	.916	.902
$e_{11}(1) = .281$	.285	.268			
$e_{12}(1) = .205$	.223	.301	$e_{11.34}(1) = .162$	.286	.269
$e_{13}(1) = .474$	.453	.385	$e_{12.34}(1) = .255$	.224	.302
$e_{14}(1) = .040$	.038	.046	$e_{13.34}(1) = .583$	.490	.429
$e_{31}(1) = .035$	.033	.032			
$e_{32}(1) = .015$	.015	.021	$e_{31.34}(1) = .043$	.035	.035
$e_{33}(1) = .860$	.862	.827	$e_{32.34}(1) = .039$	.016	.022
$e_{34}(1) = .090$	.090	.121	$e_{33.34}(1) = .918$	.949	.942

* $p_{ij.34}(1)$ is the ij-th element of $P_{34}(1)$; similarly for the residence matrix.

In table 5 we see that sample 1 happened to yield estimates that are quite close to the true values, whereas sample 10 with twice as many observations has not done as well. In practice not all of the estimates in such a table as 5 may have a useful interpretation. In our case, $e_{12}(1)$, for example, would have little practical significance.

It may be of interest to know that to compute $P(1/10)$, Q, $P(1)$, $e(1)$, $P_{34}(1)$, and $e_{34}(1)$ for a single sample took about 6 hours. A 4 state model with no *a priori* restrictions would have taken about twice as long. It took about 15 terms of the series (2.3) and (3.1) to secure convergence accurate to 5 decimal places. To compute Q took fewer terms.

APPENDIX 1

Theorem: If

$$Qt = P^*(t) - \frac{(P^*(t))^2}{2} + \frac{(P^*(t))^3}{3} - \cdots \tag{A1.1}$$

converges (elementwise) then (A1.1) is equivalent to

$$P^*(t) = Qt + Q^2\frac{t^2}{2!} + Q^3\frac{t^3}{3!} + \cdots. \tag{A1.2}$$

Proof: The coefficients of (A1.2) are the same as the coefficients of the power series representation of $e^x - 1 = y$:

$$y = x + \frac{x^2}{2!} + \frac{x^3}{3!} + \cdots, \tag{A1.3}$$

while the coefficients of (A1.1) are the same as the coefficients of the power series representation of $x = \log(1+y)$, for $|y| < 1$:

$$x = y - \frac{y^2}{2} + \frac{y^3}{3} - \cdots. \tag{A1.4}$$

First assume (A1.2) is true. Substituting (A1.2) into (A1.1) we obtain on the right side of (A1.1) the series

$$Qt + a_2(Qt)^2 + a_3(Qt)^3 + \cdots \tag{A1.5}$$

where $a_2, a_3, \cdots$ are certain constants. The series (A1.5) converges by hypothesis. Substituting (A1.3) into (A1.4) we obtain on the right side of (A1.4) the series

$$x + a_2x^2 + a_3x^3 + \cdots \tag{A1.6}$$

which has the identical coefficients as (A1.5). This follows from the fact that $P^*(t)$, Qt, x and y enter only as algebraic elements following the same multiplication and addition laws. However, since (A1.6) converges and equals x for an infinite number of values of x, all the coefficients $a_2, a_3, \cdots$ are zero. Therefore (A1.5) equals Qt and (A1.1) is true. The argument for the converse is the same given the fact that the exponential series converges for all values of the argument.

APPENDIX 2

A sufficient condition for the convergence (elementwise) of

$$\text{(A2.1)} \qquad \sum_{n=1}^{\infty} P^{*(n)}(t) \frac{(-1)^{n+1}}{n}$$

is that

$$\text{(A2.2)} \qquad p_{ii}(t) > \tfrac{1}{2} \quad \text{for} \quad i = 1, \cdots, k.$$

Proof: Denote the ij'th element of $P^{*(n)}(t)$ by $p^*{}_{ij}{}^{(n)}(t)$. (A2.2) is equivalent to $\sum_{\alpha \neq i} p_{i\alpha}(t) < \frac{1}{2}$ for $i = 1, \cdots, k$; hence $| p^*{}_{ij}{}^{(1)}(t) | < \frac{1}{2}$ for $i, j = 1, \cdots, k$. Now assume that

$$\text{(A2.3)} \qquad | p^*{}_{ij}{}^{(n)}(t) | < \tfrac{1}{2} \quad \text{for} \quad i, j = 1, \cdots, k$$

for some value of n. By definition,

$$\begin{aligned} p^*{}_{ij}{}^{(n+1)}(t) &= \sum_{\alpha=1}^{k} p^*{}_{i\alpha}(t) p^*{}_{\alpha j}{}^{(n)}(t) \\ &= \sum_{\alpha} p_{i\alpha}(t) p^*{}_{\alpha j}{}^{(n)}(t) - p^*{}_{ij}{}^{(n)}(t) \\ &= \sum_{\alpha \neq i} p_{i\alpha}(t) p^*{}_{\alpha j}{}^{(n)}(t) + (p_{ii}(t) - 1) p^*{}_{ij}{}^{(n)}(t). \end{aligned}$$

By (A2.2) and (A2.3) the absolute values of each of the last two summands is less than $\frac{1}{4}$, so that $| p^*{}_{ij}{}^{(n+1)}(t) | < \frac{1}{2}$. Hence, by induction, (A2.3) holds for all n.

Now, by (A2.2), there exists a $\delta > 1$ such that $| \delta p^*{}_{ii}(t) | < \frac{1}{2}$ for $i = 1, \cdots, k$. Substituting $\delta p^*{}_{ij}(t)$ for $p^*{}_{ij}(t)$, for all i, j, it follows from what has just been proved that

$$| \delta^n p^*{}_{ij}{}^{(n)}(t) | < \tfrac{1}{2} \quad \text{or} \quad | p^*{}_{ij}{}^{(n)}(t) | < \frac{1}{2\delta^n}$$

for $i, j = 1, \cdots, k$ and all n. Hence,

$$\sum_{n=1}^{\infty} \left| p^*{}_{ij}{}^{(n)}(t) \frac{(-1)^{n+1}}{n} \right| < \tfrac{1}{2} \sum_{n=1}^{\infty} \frac{1}{n\delta^n} \quad \text{for} \quad i, j = 1, \cdots, k.$$

The infinite sum on the right converges, and since absolute convergence implies convergence, it follows that (A2.1) converges.

APPENDIX 3

We give here the derivation of the asymptotic distributions of $\hat{p}_{ij}(t)$ and $\hat{e}_{ij}(t)$.

Observe first that (5.4) has the form

$$n \sum_{b} \frac{n}{n_b} h_b(\alpha, \beta, \gamma, \delta) = n c_{\alpha\beta\gamma\delta}(n)$$

where $n = n_1 + \cdots + n_k$ and $c_{\alpha\beta\gamma\delta}(n)$ depends on n but is bounded as $n \to \infty$. Therefore, the asymptotic covariance of $\hat{q}_{\alpha\beta}$ and $\hat{q}_{\gamma\delta}$ for $\alpha \neq \beta$ and $\gamma \neq \delta$ has the form

$$\text{(A3.1)} \qquad \frac{1}{n} c'_{\alpha\beta\gamma\delta}(n)$$

where $c'_{\alpha\beta\gamma\delta}(n)$ depends on n but is bounded. In particular $\hat{q}_{\alpha\beta}$ has the asymptotic variance $\frac{1}{n} c'_{\alpha\beta\alpha\beta}(n) = \frac{1}{n} c'_{\alpha\beta}{}^2(n)$.

Now expanding $\hat{p}_{ij}(t)$ in a Taylor expansion about the $q_{\alpha\beta}$'s, we obtain

$$\text{(A3.2)} \qquad \sqrt{n}(\hat{p}_{ij}(t) - p_{ij}(t)) = \sqrt{n} \sum_{\alpha \neq \beta} \left(\frac{\partial p_{ij}(t)}{\partial q_{\alpha\beta}} \right) (\hat{q}_{\alpha\beta} - q_{\alpha\beta}) + \sqrt{n} R.$$

The remainder R is the quadratic form

$$\text{(A3.4)} \qquad \sum_{\alpha \neq \beta} \sum_{\gamma \neq \delta} \left(\frac{\partial^2 p'_{ij}(t)}{\partial q'_{\alpha\beta} \partial q'_{\gamma\delta}} \right) (\hat{q}_{\alpha\beta} - q_{\alpha\beta}) (\hat{q}_{\gamma\delta} - q_{\gamma\delta}),$$

where the derivatives are evaluated at points $q'_{\alpha\beta}, q'_{\gamma\delta}, \cdots$ intermediate to $\hat{q}_{\alpha\beta}$ and $q_{\alpha\beta}$, $\hat{q}_{\gamma\delta}$ and $q_{\gamma\delta}, \cdots$ respectively.

We show first that $\sqrt{n}R$ converges in probability to zero. Since $\hat{q}_{\alpha\beta}$ is asymptotically normally distributed, we have

$$\text{(A3.5)} \qquad \Pr\{ | \hat{q}_{\alpha\beta} - q_{\alpha\beta} | \geq \frac{1}{\sqrt{n}} c'_{\alpha\beta}(n) u \} \to 2(1 - \Phi(u))$$

where $\Phi(u)$ is the unit normal distribution function. Let ϵ and δ be given such that $1 > \epsilon > 0$, and $1 > \delta > 0$. First choose u so large that

$$2(1 - \Phi(u)) < \frac{\delta}{k(k-1)} \tag{A3.5}$$

(where k is the size of the Q matrix). Then choose n' such that for $n > n'$ all the inequalities

$$\frac{1}{\sqrt[4]{n}} c'_{\alpha\beta}(n) u < \epsilon \qquad \alpha \neq \beta \tag{A3.7}$$

are satisfied simultaneously. Finally, for each n, define Z_n as the set of sample points for which all the inequalities

$$| \hat{q}_{\alpha\beta} - q_{\alpha\beta} | < \frac{1}{\sqrt{n}} c'_{\alpha\beta}(n) u \qquad \alpha \neq \beta \tag{A3.8}$$

are satisfied simultaneously, and define Z^*_n as the complementary set.

It follows from (A3.5) and A3.6) that for all n greater than some n'', say,

$$\Pr(Z^*_n) < k(k-1) \frac{\delta}{k(k-1)} = \delta$$

and hence $\Pr(Z_n) \geq 1 - \delta$ for $n > n''$. But on Z_n it follows from (A3.7) and (A3.8) that for $n > n'$,

$$\sqrt{n} | \hat{q}_{\alpha\beta} - q_{\alpha\beta} | \, | \hat{q}_{\gamma\delta} - q_{\gamma\delta} | < \frac{1}{\sqrt{n}} c'_{\alpha\beta}(n) c'_{\gamma\delta}(n) u^2$$

$$< \frac{1}{\sqrt{n'}} c'_{\alpha\beta}(n) c'_{\gamma\delta}(n) u^2 < \epsilon^2.$$

Since ϵ and δ are arbitrarily close to zero, it follows that the term on the left converges in probability to zero, and hence $\sqrt{n}R$, a linear combination of such terms, also converges in probability to zero.

Now consider $\sqrt{n} | \hat{q}_{\alpha\beta} - q_{\alpha\beta} |$. We have

$$\Pr\{ \sqrt{n} | \hat{q}_{\alpha\beta} - q_{\alpha\beta} | \geq c'_{\alpha\beta}(n) u \} \to 2(1 - \Phi(u)).$$

As $u \to 0$, $2(1 - \Phi(u)) \to 1$, so that $\sqrt{n} | \hat{q}_{\alpha\beta} - q_{\alpha\beta} |$ does not converge in probability to zero.

It follows, cancelling $\sqrt{n}$, that $(\hat{p}_{ij}(t) - p_{ij}(t))$ has the same asymptotic distribution as

$$\sum_{\alpha \neq \beta} \left(\frac{\partial p_{ij}(t)}{\partial q_{\alpha\beta}} \right) (\hat{q}_{\alpha\beta} - q_{\alpha\beta}).$$

This is a linear combination of asymptotically normal variables, hence is itself asymptotically normal, with the asymptotic mean 0 and asymptotic variance

$$\frac{1}{n}\sum_{\alpha\neq\beta}\sum_{\gamma\neq\delta}\left(\frac{\partial p_{ij}(t)}{\partial q_{\alpha\beta}}\right)\left(\frac{\partial p_{ij}(t)}{\partial q_{\gamma\delta}}\right)c'_{\alpha\beta\gamma\delta}(n).$$

For t', (the interval between successive observations) sufficiently small, this is approximately

$$\sum_{\alpha\neq\beta}\left(\frac{\partial p_{ij}(t)}{\partial q_{\alpha\beta}}\right)^2\frac{q_{\alpha\beta}}{\sum_b n_b e_{b\alpha}(T)}.$$

The asymptotic distribution of the $\hat{e}_{ij}(t)$'s is derived similarly. $\hat{e}_{ij}(t)$ is asymptotically normal with asymptotic mean $e_{ij}(t)$ and asypmtotic variance

$$\frac{1}{n}\sum_{\alpha\neq\beta}\sum_{\gamma\neq\delta}\left(\frac{\partial e_{ij}(t)}{\partial q_{\alpha\beta}}\right)\left(\frac{\partial e_{ij}(t)}{\partial q_{\gamma\delta}}\right)c'_{\alpha\beta\gamma\delta}(n).$$

For t' sufficiently small this is approximately

$$\sum_{\alpha\neq\beta}\left(\frac{\partial e_{ij}(t)}{\partial q_{\alpha\beta}}\right)^2\frac{q_{\alpha\beta}}{\sum_b n_b e_{b\alpha}(T)}.$$

SUMMARY

The general, time stationary, continuous, Markov process with a finite number of states is introduced as a model for a follow-up study. This model may prove useful in situations in which several states of the disease can be distinguished and are of clinical importance, as opposed to just two states, life, and death. A special case of this process was used by Fix and Neyman ('51) for a 4 state model.

The estimate of the parameters of the model, called transition rates, is based on observations of patients at equal intervals of time t', for a total period of time $st' = T$. Letting $P(t')$ be the matrix of transition probabilities for $t = t'$, and $\hat{P}(t')$ the maximum likelihood estimate of $P(t')$, the matrix Q of transition rates is estimated by

$$\hat{Q} = \log\hat{P}(t') = (\hat{P}(t') - I) - (\hat{P}(t') - I)^2/2 + (\hat{P}(t') - I)^3/3 - \cdots,$$

provided the series converges. A simple condition for convergence is that the diagonal elements of $\hat{P}(t') - I$ be less than $\frac{1}{2}$.

The asymptotic distributions of the elements of $\hat{Q}$, $\hat{P}(t)$, and

$\hat{e}(t) = \int_0^t \hat{P}(u)du$, the residence matrix, are all normal. The effect of letting $t' \to 0$, upon the asymptotic variance of the elements of $\hat{Q}$ is examined in a special case. When t' is small the formulas simplify considerably.

Some tests of hypotheses concerning the equality of transition rates, transition probabilities, and residencies (elements of $e(t)$), and some goodness of fit tests of the model are given. The model is extended to two (or more) contiguous periods, each with separate transition rate matrices. Finally the results of a sampling experiment using a 4 state model are given.

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NOTE ON ESTIMATION IN A MARKOV PROCESS WITH CONSTANT TRANSITION RATES *

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REMARKS

IN the foregoing paper Mr. Zahl presents as a model for follow-up studies the general Markov process with a finite number of states, say $S_1, \cdots, S_k$, and constant transition rates, q_{ij} = transition rate from S_i to S_j. As he remarks, special cases have been examined before, notably by Fix and Neyman ('51). Indeed, perhaps the simplest case of this process is just the familiar exponential survivorship (or constant death rate) model so often used both in medical and industrial applications.

In both the Fix-Neyman paper and in the foregoing excellent study, observation is assumed to be incomplete—the status of each subject is known only at certain times during the follow-up interval. Although such restrictions are clearly appropriate to many applications, there are others in which transitions from one state to another are sufficiently dramatic (*e.g.*, living to dead) that observation may be regarded as essentially complete over the follow-up interval. Since, also, the maximum likelihood estimates of the basic parameters, q_{ij}, are exceedingly simple when there is complete information, it seems appropriate to introduce them along with the general stochastic model.

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THE ESTIMATES

Briefly, q_{ij} is the constant rate of transition from state S_i to state S_j and the maximum likelihood estimate of q_{ij} is simply

$$\hat{q}_{ij} = \frac{m_{ij}}{T_i},$$

where

$m_{ij} =$ total transitions observed from S_i to S_j, summed over all subjects,

$T_i =$ total time under observation in state S_i, summed over all subjects.

This is a well known result for the special case of exponential survivorship mentioned earlier (Littell, '52, and Epstein and Sobel, '53) in which we have one transient state (surviving) and one absorbing state (dead or failed). The extension of the derivation to the general case is straightforward and will not be discussed here.

AN EXAMPLE

Consider a model for chronic disease follow-up with three states, S_1, S_2, S_3, representing the states "well," "sick," and "dead." (S_1 and S_2 are "transient" states and S_3 is an "absorbing" state.) Suppose the study involves only two individuals starting in S_2, with the following histories. The first becomes well at 1.4 years, relapses at 2.3 years, regains health at 3.2 years and remains well for the rest of the 5-year follow-up interval. The second becomes well at 0.8 years, relapses at 1.6 years, and dies at 3.0 years. The data may be tabulated as shown in table 1.

TABLE 1

CASE 1		CASE 2	
state	period	state	period
S_2	0.0 to 1.4	S_2	0.0 to 0.8
S_1	1.4 to 2.3	S_1	0.8 to 1.6
S_2	2.3 to 3.2	S_2	1.6 to 3.0
S_1	3.2 to 5.0	S_3	3.0 to 5.0

Since S_3 is an absorbing state, $m_{31} \equiv m_{32} \equiv 0$, and T_3 is not needed. The required quantities are

$$\begin{aligned} T_1 &= [(2.3 - 1.4) + (5.0 - 3.2)] + [(1.6 - 0.8)] \\ &= 2.7 + 0.8 = 3.5 \end{aligned}$$

$$\begin{aligned} T_2 &= [(1.4 - 0.0) + (3.2 - 2.3)] + [(0.8 - 0.0) + (3.0 - 1.6)] \\ &= 2.3 + 2.2 = 4.5 \end{aligned}$$

$$m_{12} = 1 + 1 = 2 \qquad m_{13} = 0 + 0 = 0$$

$$m_{21} = 2 + 1 = 3 \qquad m_{23} = 0 + 1 = 1$$

The estimates of the transition rates are

$$\hat{q}_{12} = 2/3.5 = 0.571 \qquad \hat{q}_{13} = 0/8.1 = 0.000$$

$$\hat{q}_{21} = 3/4.5 = 0.667 \qquad \hat{q}_{23} = 1/4.5 = 0.222.$$

DISTRIBUTION OF ESTIMATES

One's intuition about the distribution of these estimates may be sharpened by examining some methods for generating a random observation from any given process. The method indicated in Doob ('53) is as follows. A subject starting in state S_{i_0} remains there for a period of time randomly chosen from an exponential distribution with mean $1/q_{i_0}$ $(q_{i_0} = \sum_{j \neq i_0} q_{i_0 j})$. He is then transferred to a state S_{i_1}, chosen at random from the states other than S_{i_0}, the chance of going to S_j being $q_{i_0 j}/q_{i_0}$ $(j \neq i_0)$. The subject then remains in state S_{i_1} a time randomly chosen from an exponential distribution with mean $1/q_{i_1}$ and then is transferred to a state different from S_{i_1} and so on. An equivalent method, suggested by the notion of competition among the states, is to choose $k - 1$ times at random from exponential distributions with means $1/q_{i_0 j}$ $(j \neq i_0)$ and make a transfer to that state, S_{i_1}, for which the chosen time was least, making the transition at this minimum time. (The needed random observations from exponential distributions can be generated from random digits, utilizing the fact that if x is uniformly distributed in the interval $(0, 1)$, $-q \ln x$ has an exponential distribution with mean q.)

The exact distribution of the q_{ij} is not at all a simple matter. If they could be regarded as being generated by *fixed and predetermined* total observation times, T_i, the m_{ij} would have independent Poisson distributions and the $\hat{q}_{ij}$ would have mean q_{ij} and variance q_{ij}/T_i, giving a coefficient of variation of $\dfrac{\sqrt{q_{ij}/T_i}}{q_{ij}} = \dfrac{1}{\sqrt{q_{ij} T_i}} = \dfrac{1}{\sqrt{E\{m_{ij}\}}}$. In fact, the likelihood function does not distinguish between the cases of fixed and random T_i so that the asymptotic formula for the variance of $\hat{q}_{ij}$, derived in the usual way, is just q_{ij}/T_i. Unfortunately the T_i *are* random variables, and the above results for the mean and variance are therefore only approximate. If, however, T_i is large compared to the mean time required for one transfer from S_i to S_j, that is $1/q_{ij}$, the effect of its variability should be slight. Since T_i must be many times $1/q_{ij}$ for $\hat{q}_{ij}$ to have even modest precision, it seems reasonable to expect that the asymptotic Poisson theory will give good service in the cases of most interest, that is, when the data provide reasonably precise estimates of the q_{ij}.

Although the hypothetical study used in the example is far too small to provide useful estimates, it will serve to exhibit the method of calculation of the variance estimates. Letting $\hat{\sigma}_{ij}^2 = \hat{q}_{ij}/T_i$, the estimated variance of $\hat{q}_{ij}$, we have

$$\hat{\sigma}_{12}^2 = \frac{0.571}{3.5} = 0.163 \qquad \hat{\sigma}_{13}^2 = \frac{0.000}{3.5} = 0.000$$

$$\hat{\sigma}_{21}^2 = \frac{0.667}{4.5} = 0.148 \qquad \hat{\sigma}_{23}^2 = \frac{0.222}{4.5} = 0.049.$$

The estimated coefficients of variation are given by $1/\sqrt{m_{ij}}$ and range from 58% for $\hat{q}_{21}$ to infinity for $\hat{q}_{31}$, showing the inadequacy of such a sample for estimating rates.

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LONGEVITY OF RETIRED ARMY AND AIR FORCE OFFICERS *

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INTRODUCTION

DEMOGRAPHERS, actuaries, and vital statisticians have long used average expectation of life as a measure of mortality and of the extent to which man has gained control over his biological environment. Accompanying the rapid industrialization of our economy and the emergence of a distinct labor force in recent years, life table principles have also been applied to computations of average length of working life. Partially as a result of the demographic, economic, and social changes of the last century and particularly of the last 50 years, the number of persons reaching retirement age has increased tremendously and there is now a considerable span of life for millions of persons between the date of retirement and death. This particular study is focused upon the computation of life expectancy after working life, that is, with measuring length of life in a retired status. Few life tables have been constructed for retired populations.

The life table applied to retired populations presents certain problems, both of construction and interpretation. The several values used in its computation can, however, be profitably utilized to: (a) predict future deaths in a retired population for a reasonable period of time,

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* Personal views or opinions expressed or implied in this article are not to be construed as necessarily carrying the official sanction of the Department of the Air Force or of the Air Research and Development Command.

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The authors are indebted to Jerry Walker Combs, Jr., for critically reading the manuscript and for valuable suggestions.

(b) measure the man-years of life remaining for retired individuals—an index of manpower potential, and (c) provide a stationary population for comparative purposes. The latter is perhaps the most limited in utility, and both (a) and (b) above must be used with due regard for the time reference of the life table functions and the structural peculiarities of the actual retired population.

MATERIALS AND METHODS

Selection of the Method Used in This Study

The retired population which is the concern of this study was composed of 10,001 United States Army (and Air Force) officers living in retirement between January 1, 1925 and December 31, 1948. From this group, three were omitted for lack of complete information, leaving 9,998 for analysis. Of the latter, 1,906 were living in retirement at the beginning of the period, and 8,092 retired on or after January 1, 1925. During the 24-year period there were 2,795 deaths, 1,509 of which occurred among officers retiring during the period. Just above 7,000 (7,203) were therefore living in retirement on December 31, 1948.

The primary consideration for the selection of the methodology used was that the population was small and changing rapidly in both size and composition. Because of the small number and limited period of time covered, a cohort life table could not be constructed (Jaffe, '51, p. 3). Such a table would at any rate have had the added disadvantage of the varying ages at which individuals entered the population and the purely historical interest of the mortality experience reflected.[1] On the other hand, the retired population at any one time was too small to allow the construction of a current life table by the usual method of using a mid-point population and a three-year average of deaths.

The method selected may perhaps be classified as a current multiple decrement table, but certain departures in selection of population and use of deaths have been made (Dublin *et al.*, '49, pp. 279-280 and U. S. Dept. of Labor, '50). Instead of being based on a specified calendar year, the rates computed are for a hypothetical "average" year in this period. Each of the retired officers has been counted in the population at each year of age or portion of year during which his life experience could be observed. Thus, most individuals entered into several age groups as a result of aging during the 24-year span. Conceivably an officer could have entered into the base population of 24 different age groups. In actuality, the average number was 7 or 8.[2] Thus the population for any year of age consisted of all officers in retirement who attained that age during the period and all officers who retired at that age. The 50-year age group, for example, consisted of all officers who retired at age 50 or attained age 50 in retirement during the 24-year period. The 51-year age group consisted of all officers who survived their fiftieth year, plus those who retired at age 51. In general, since additional retirements occurred as age increased, and because officers retiring at younger ages grew older during the period, the population used for computing age-specific rates generally increased up to age 64. It was only after that age that attrition through death outweighed the increments to the population.

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[1] Observed survival ratios have been computed, however, for those who were in a retired status when the study began as well as for those who retired during the period 1925-1929.

Obviously, it was not possible to treat the population arrived at as an enumerated population. It was possible, however, to follow two courses. The population could have been computed on the basis of the total number attaining to each x birthday in retirement, making possible the direct computation of the probability of dying; or it was possible to convert the population into years lived in retirement at age x, making possible the computation of the death rate. Because the former would require sacrificing certain data and would involve complications in making the computations by 5-year age groups, and because of certain advantages in age-specific death rates themselves, it was decided to compute the death rate and to deal with the population on a years-lived (years of exposure) basis.

Accordingly, the ${}_nm_x$ (a convenient summary of the conventional notation used can be found in Reed and Merrell, '39) for any given age x was computed from $\frac{{}_nD_x}{{}_nL'_x}$ rather than from the more usual $\frac{{}_nD_x}{{}_nP_x}$.[3] The ${}_nD_x$ included all of the deaths for an age group occurring during the 24-year span, the ${}_nL'_x$ representing the years lived (observed) in retirement in the interval x to $x+n$. It is immediately apparent that the formula ${}_nm_x = \frac{{}_nD_x}{{}_nL'_x}$ is roughly equivalent to deriving the ${}_nm_x$ from the $\frac{{}_nd_x}{{}_nL_x}$ values of a life table (${}_nd_x$ = deaths in the life table age group x to $x+n$; ${}_nL_x$ = life table population in the age group x to $x+n$). Theoretically it may be considered a more accurate value for ${}_nm_x$ than that usually employed, but it is perhaps influenced at ages up to 65 by the fact that increments to the population occurred during a year of life, so that both the numerator and denominator are expanding values during a year of life. The theoretical consequences of this fact have not been thoroughly explored. They undoubtedly have some bearing on the convertibility of the ${}_nm_x$ values to ${}_nq_x$ (probability of dying) values by the Reed and Merrell method.

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[2] Computed by dividing total man-years lived during the period by the maximum population, 9,998. This gives the average years lived in retirement.

[3] Deaths in the age group x to $x + n = {}_nD_x$; population in the age group x to $x + n = {}_nP_x$. For convenience, the observed person-years lived at each age has been denoted by ${}_nL'_x$. Thus the death rate ${}_nm_x$ really amounts to deaths per 1,000 years of exposure.

The methods used in actual computations were those adaptable to (1) data available and (2) IBM tabulation.[4]

Available Data

The following data were available for use in the study of length of life of retired officers (Official Registers, '25-'49):

Recorded data. (1) Date of birth of each officer, (2) date of retirement of each officer, (3) date of death of each officer who died, (4) date of beginning of study (January 1, 1925), (5) date of termination of study (December 31, 1948).

Computed data. (1) Age at retirement of each officer, (2) age as of December 31, 1924 for those retiring previous to January 1, 1925, (3) age at death of each officer who died, (4) age as of December 31, 1948 for those officers alive on that date. (All ages were computed in years to two decimal places.) The various possibilities of contributions to the years lived in retirement were listed and are shown in figure 1.

Years Lived in Retirement (Years of Exposure)

The years lived in retirement were computed by the method illustrated in table 1; this method allowed a detailed check of the data at a

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[4] The authors are indebted to Stephen J. Zolczynski for furnishing the IBM methodology; they are indebted to Stephen W. Fotis for providing a manual verification on a sample basis of the method used.

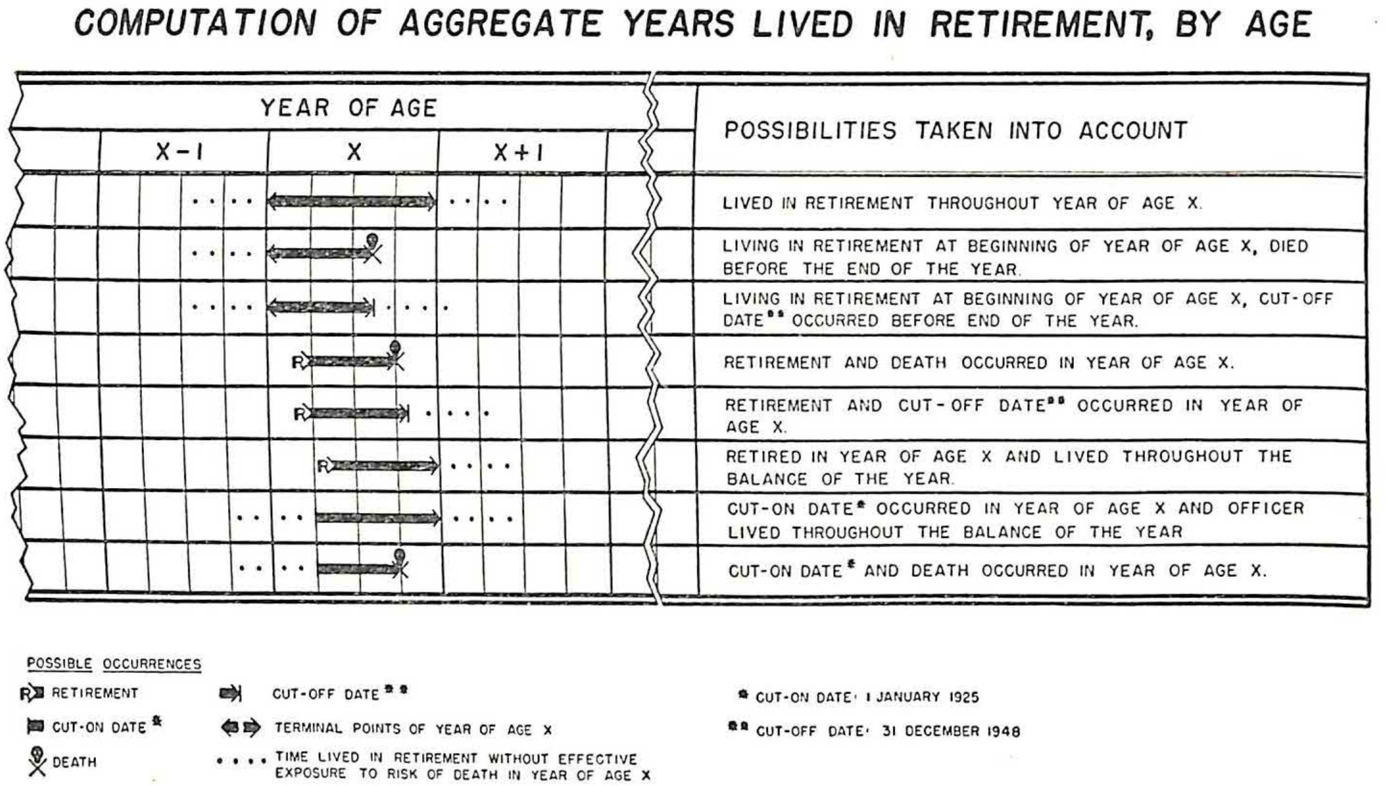
COMPUTATION OF AGGREGATE YEARS LIVED IN RETIREMENT, BY AGE
YEAR OF AGE
X-1
X
X+1
POSSIBILITIES TAKEN INTO ACCOUNT
LIVED IN RETIREMENT THROUGHOUT YEAR OF AGE X.
LIVING IN RETIREMENT AT BEGINNING OF YEAR OF AGE X, DIED BEFORE THE END OF THE YEAR.
LIVING IN RETIREMENT AT BEGINNING OF YEAR OF AGE X, CUT-OFF DATE** OCCURRED BEFORE END OF THE YEAR.
RETIREMENT AND DEATH OCCURRED IN YEAR OF AGE X.
RETIREMENT AND CUT-OFF DATE** OCCURRED IN YEAR OF AGE X.
RETIRED IN YEAR OF AGE X AND LIVED THROUGHOUT THE BALANCE OF THE YEAR.
CUT-ON DATE* OCCURRED IN YEAR OF AGE X AND OFFICER LIVED THROUGHOUT THE BALANCE OF THE YEAR
CUT-ON DATE* AND DEATH OCCURRED IN YEAR OF AGE X.
R
POSSIBLE OCCURRENCES
RETIREMENT
CUT-ON DATE*
DEATH
CUT-OFF DATE**
TERMINAL POINTS OF YEAR OF AGE X
. . . . TIME LIVED IN RETIREMENT WITHOUT EFFECTIVE EXPOSURE TO RISK OF DEATH IN YEAR OF AGE X
* CUT-ON DATE: 1 JANUARY 1925
** CUT-OFF DATE: 31 DECEMBER 1948

FIG. 1. POSSIBILITIES TAKEN INTO ACCOUNT IN COMPUTING PERSON-YEARS LIVED IN RETIREMENT BY RETIRED OFFICERS OF THE UNITED STATES ARMY AND AIR FORCE, 1925-1948.

glance and yet the machine process was simple.[5] Figure 2 shows the "population" (years of exposure) obtained.

Age Specific Death Rates

Having secured the population it was only necessary to compute ${}_nm_x$ by the formula $\frac{{}_nD_x}{{}_nL'_x}$, as previously given, ${}_nL'_x$ denoting the number of years lived in the interval x to $x+n$. As stated previously, an approximate equivalence between $\frac{{}_nD_x}{{}_nP_x}$, $\frac{{}_nD_x}{{}_nL'_x}$, and $\frac{{}_nd_x}{{}_nL_x}$ has been assumed, although each implies a different type of base population. The ${}_nm_x$ values for the entire retired population, compared with those of white

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[5] As the first step in the computation process (IBM), the population was ordered by age at retirement. The next step was to cumulate for each age the number of officers who lived in retirement at that age. Each person who retired was counted as contributing a year at the age of retirement and each succeeding year through age 103. (Age 103 was taken as the final age since no officer in the population lived beyond that age during the period observed. The maximum population of 9,998 was reached at age 94 when the last person had retired.) This gave the total number of retirement years that would have been contributed at each age if (a) a person contributed a full year at the age which he entered the population and (b) no persons left the population. Since these two conditions did not hold, appropriate corrections had to be made. To correct for the fact that few or no officers actually contributed a full year in the year of entry, the actual proportion of the year which they did contribute was computed. The machine technique selected for doing this was to compute and tabulate the proportion of the year which was not contributed. Then by a clerical operation this amount was subtracted from the full year which had been credited at age of entry.

A similar technique was used for subtracting dropouts. Since an officer was credited with a full year for every age after he had entered the population, it was necessary to subtract a full year for every age after he had died and for all ages which would have been attained after December 31, 1948. This was a matter of accumulating the number of dropouts from age 23 onward and subtracting the cumulative total at each age. For the age at which an individual was lost to the population, however, a portion of the year was actually lived in retirement. Since he was already credited with contributing a full year at that age the portion of that year not lived in retirement was subtracted as a correction factor.

In summary, the method was to reckon the maximum retirement years that an officer could contribute and then to subtract all the full years and fractions of years that he was known not to have contributed. Two IBM machine runs only were required, the final number of years lived at each age being obtained by simple subtraction.

TABLE 1

Example of the Computations Used to Obtain Person-Years Lived in Retirement by Retired Officers of the United States Army and Air Force, 1925-1948

	ACCESSIONS				SEPARATIONS				NET
Year of age (1)	Number of persons retiring (IBM) (2)	Cumulative increments—maximum possible person-years of retirement (IBM) (3)	Non-contributive person-years of increments (IBM) (4)	Contributive potential person-years of retirement (3)-(4) (5)	Number of persons dropping out of retired population (IBM) (6)	Cumulative decrements—maximum possible person-years lost through decrements (IBM) (7)	Contributive person-years of decrements (IBM) (8)	Non-contributive years of decrements (7)-(8) (9)	Person-years lived in a retired status (5)-(9) (10)
22	3	3	2.54	.46	—	—	—	—	.46
23	3	6	1.09	4.91	1	1	.48	.52	4.39
24	12	18	6.60	11.40	1	2	.20	1.80	9.60
. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .
90	1	9993	.02	9992.98	17	9953	7.58	9945.42	47.56
91	1	9994	.81	9993.19	9	9962	3.84	9958.16	35.03
92	3	9997	1.33	9995.67	17	9979	7.79	9971.21	24.46
93	1	9998	.38	9997.62	3	9982	1.32	9980.68	16.94
94	—	9998	—	9998.00	3	9985	1.74	9983.26	14.74
95	—	9998	—	9998.00	3	9988	1.46	9986.54	11.46
96	—	9998	—	9998.00	3	9991	1.44	9989.56	8.44
97	—	9998	—	9998.00	3	9994	2.05	9991.95	6.05
98	—	9998	—	9998.00	1	9995	.19	9994.81	3.19
99	—	9998	—	9998.00	—	9995	—	9995.00	3.00
100	—	9998	—	9998.00	2	9997	1.63	9995.37	2.63
101	—	9998	—	9998.00	—	9997	—	9997.00	1.00
102	—	9998	—	9998.00	1	9998	.36	9997.64	.36
103	—	9998	—	9998.00	—	9998	—	9998.00	—

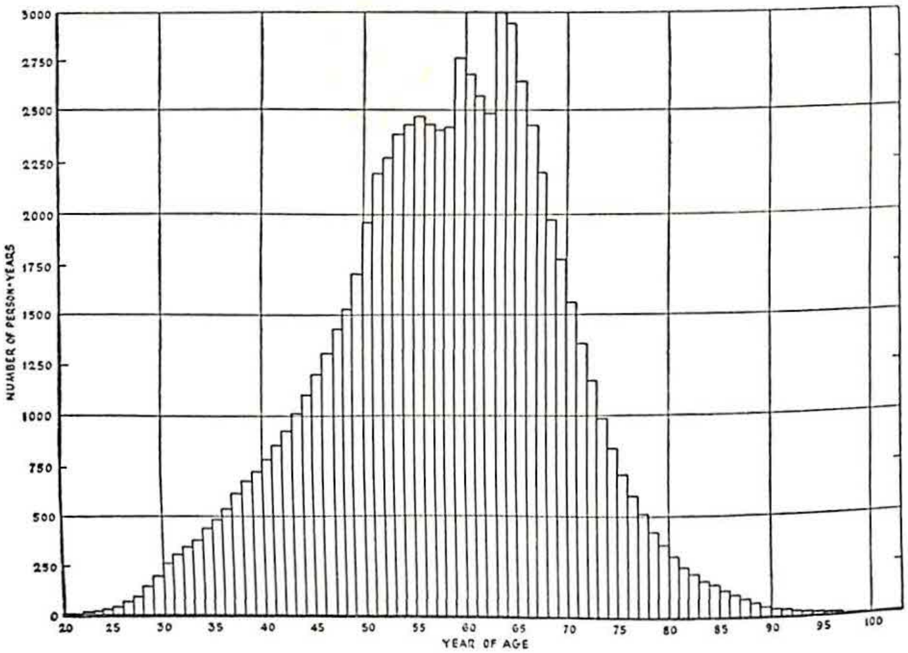

FIG. 2. PERSON-YEARS LIVED IN RETIREMENT BY RETIRED OFFICERS OF THE UNITED STATES ARMY AND AIR FORCE, 1925-1948.

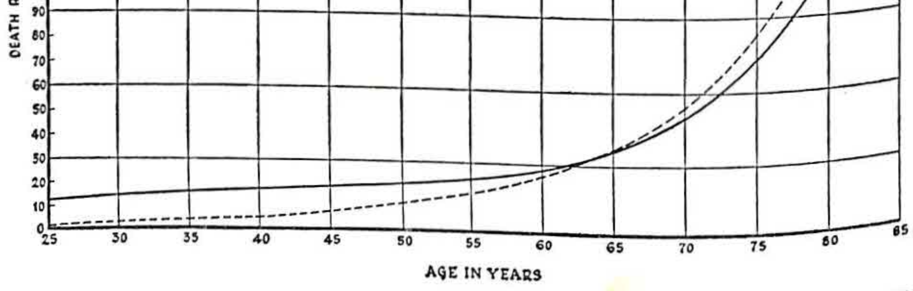

FIG. 3. AGE SPECIFIC DEATH RATES OF WHITE MALES OF THE UNITED STATES, 1939-1941, COMPARED WITH THOSE OF RETIRED OFFICERS OF THE UNITED STATES ARMY AND AIR FORCE, 1925-1948.

males in the United States 1939-1941, are shown in figure 3. The $_{n}m_{x}$ values computed for specified portions[6] of the retired population are given in figure 4.

Smoothing the data. The smooth curves of mortality applying to retired officers shown in figures 3 and 4 are, of course, not the actual observed rates obtained. Despite the inflation of the population through computing years of exposure, the population at all ages was still extremely small. A total of 10,001 individual officers constituted the initial population, of whom three had to be eliminated because of lack of data. Thus 9,998 officers lived in a retired status averaging less than 8 years each. The total population (person-years lived) at all ages was therefore less than 80,000, actually 76,172.81. Against these there were only 2,795 deaths. The resulting mortality rates were therefore highly irregular. On the usual assumption that these irregularities are not a feature of the "true" mortality rate, they have been graduated mechanically. In this case it appeared that any mathematical formula would be as arbitrary a method of smoothing as drawing the curve with the aid of a spline.

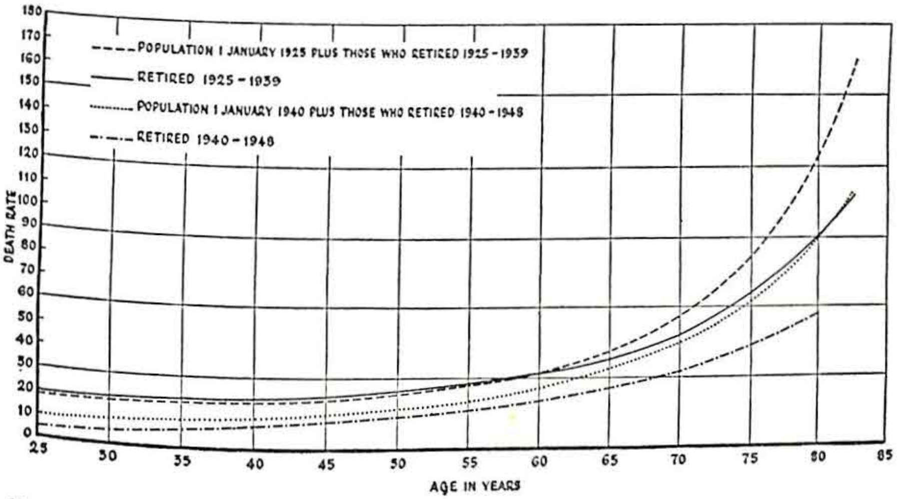

FIG. 4. AGE-SPECIFIC DEATH RATES OF SUB-GROUPS OF THE RETIRED OFFICER POPULATION OF THE UNITED STATES ARMY AND AIR FORCE, 1925-1948.

The resultant curves were tested for smoothness by the method of third differences. The results compared favorably with Greville's test of white males for 1939-1941 (Greville, '46, pp. 126-129). In addition, the graduated rates were applied to the actual years of exposure and produced the results shown in table 2. These were considered satisfactory.

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[6] Computations for these sub-groups were made in the manner previously described by means of additional "cut-on" and "cut-off" dates.

TABLE 2

Actual Deaths Compared to Number of Deaths Produced by Smoothed Data for Selected Age Groups of Retired Officers of the United States Army and Air Force, 1925-1948

AGE GROUP	OBSERVED DEATHS	SMOOTHED DATA
30–39	67	71
40–49	230	210
50–59	549	565
60–69	915	924
70–79	685	685
80–89	288	289
Total 30–89	2734	2739

Probability of Dying

Conversion of the computed ${}_nm_x$ values to ${}_nq_x$ by means of the formula $q_x = \frac{2m_x}{2 + m_x}$ was explored but was discarded in favor of the widely-used Reed and Merrell tables ('39). One possible limitation should be recognized here. Since the Reed and Merrell tables give the relationship of ${}_nm_x = \frac{{}_nD_x}{{}_nP_x}$ to ${}_nq_x = \frac{{}_nd_x}{{}_nl_x}$, it has been assumed in this study that the same relationship holds when ${}_nm_x = \frac{{}_nD_x}{{}_nL'_x}$. Some question regarding the equivalence of the two formulas for ${}_nm_x$ has already been raised above, but the Reed and Merrell tables appear more satisfactory for all ranges of ages than the formula $\frac{2m_x}{2 + m_x}$.

RESULTS

The findings of the present study are presented in figures 3 and 4, and in table 3. For the retired population, age-specific rates were higher at ages under 65 than were rates for corresponding ages among the white male population in 1939-1941. Above age 65, however, rates were lower for the retired officer population (figure 3). The higher

TABLE 3

Abridged Life Table for all Retired Officers of the United States Army and Air Force, 1925-1948 (Constructed by Reed and Merrell Method)

AGE INTERVAL	PROPORTION DYING	OF 100,000 RETIRING		STATIONARY POPULATION		AVERAGE REMAINING LIFETIME
Period of life between two exact ages stated in years (1)	Proportion of persons alive at beginning of age interval dying during interval (2)	Number alive at beginning of age interval (3)	Number dying during age interval (4)	In the age interval (5)	In this and all subsequent age intervals (6)	Average number of years of life remaining at beginning of age interval (7)
x to $x+n$	${}_nq_x$	l_x	${}_nd_x$	${}_nL_x$	T_x	$\mathring{e}_x$
25–29	0.06732	100,000	6,732	505,430	3,493,786	34.9
30–34	.07340	93,268	6,846	449,270	2,988,356	32.0
35–39	.08036	86,422	6,945	414,757	2,539,086	29.4
40–44	.08682	79,477	6,900	380,119	2,124,329	26.7
45–49	.09461	72,577	6,867	345,688	1,744,210	24.0
50–54	.10279	65,710	6,754	311,678	1,398,522	21.3
55–59	.11761	58,956	6,934	277,599	1,086,844	18.4
60–64	.14397	52,022	7,490	241,684	809,245	15.6
65–69	.18796	44,532	8,370	202,119	567,561	12.7
70–74	.25806	36,162	9,332	157,791	365,442	10.1
75–79	.36752	26,830	9,861	109,332	207,651	7.7
80–84	.50345	16,969	8,543	62,603	98,319	5.8
85–89	.66606	8,426	5,612	26,777	35,716	4.2
90–94	.77924	2,814	2,193	7,529	8,939	3.2
95–99	.86327	621	536	1,410	1,410	2.3
100–over	—	85	—	—	—	—

mortality for retired officers at younger ages probably reflects (1) the earlier base period for the retired officer rates and (2) the "reasonably expected" lesser longevity of officers retiring at younger ages (Reed and Love, '31). How much of the difference can be attributed to the latter is not known, but there seems to be rather clear evidence that the time reference has influenced the difference. In figure 4 it can be observed that mortality rates are lower for officers retiring recently than for those retiring earlier. The factors effecting an increasing longevity for the general population in recent years thus appear also to be operative among this retired population.

DISCUSSION

When the age-specific death rates are carried forward in the life table to statements of life expectancy, a certain fiction is inevitable. All life tables incorporate such a fiction, since the stationary population is never identical with the actual population and the mortality rates are rarely applicable over the entire lifetime of any actual cohort. The present life table population, however, departs from the actual in rather extreme degree. To what extent this departure results in unreasonable life expectancies at the younger ages, however, is not known. The life expectancy of persons retiring at age 25–29 and earlier has been determined not only by their experience, but by those retiring at older ages. If those retiring at younger ages enjoy materially less longevity than those who retire at older ages, then the life expectancy value for age 25–29 probably misrepresents what could be expected from limiting analysis to this group alone.[7] In projections of total years lived in retirement the values should, of course, be obtained from actual data on persons who retire at such young ages. The life table population is manifestly not appropriate for such purposes. Useful projections may be made, however, by relating the average life expectancy at each age to the number of officers who retire at those ages. As long as the life table values may be considered as approximately applicable to a retired population, estimates of cost or of total years lived in retirement by a retired population may be obtained.

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[7] It has been possible to observe 8 individuals retiring at ages 25–29 for a period of 20 years. At the end of that time, 6 were still alive, two had died.

CONCLUSIONS

The technique of life table construction utilized in this study of retired officers might be profitably applied to many groups. For example, in the face of the current teacher shortage, a life table could aid in determining the additional personnel that would be available if the legal age of retirement were delayed five years. It should be realized however that this particular method of life table construction has both advantages and disadvantages. Its chief advantages are as follows: (1) It is an application of a method of life table construction to a relatively small and changing population that can be observed over a period of years when no cohort for a single year is large enough to warrant the construction of a conventional current table or can be observed long enough to make the construction of a cohort life table feasible. (2) It utilizes an uncomplicated machine technique which greatly simplifies the computational procedures. Its limitations, however, must be admitted. The mortality experience is not so recent as that used in strictly current life tables, but is less obsolescent than that used in cohort life tables. The farther the mid-point of the period from the current date, the less predictive value the life table will likely have. Finally, retired populations of the present type have a greater range of ages at which individuals enter the population than do most populations for which life tables have been constructed. Retired officer populations may be more extreme in this regard than other retired populations. Wherever similar populations are encountered, however, average life expectancy values must be interpreted with care.

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THE HEIGHTS AND WEIGHTS OF MEN ACCORDING TO MONTH OF BIRTH

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AT the time my book on seasonal fluctuations (Fitt, '41) was written, no data had been forthcoming to test the relationship of height and weight of adults to month of birth. Many studies were available of children of different ages. At about the same time two articles appeared offering measurements of American university and college students in a similar kind of analysis. One was by Mills ('41) and the other by Chenoweth and Canning ('41). The trends noted were in many respects similar to those of the author's analysis but not identical. It must be noted that college students represent a highly selected population, not calculated to reveal the growth condition of the population as a whole. In any case they do not represent full adult growth. The height and weight records were taken for 16-, 17-, 18- and 19-year-old freshmen who were still growing (and at different rates).

In order to secure data for a fully adult population it was decided to analyse the heights and weights of a large sample of the men of the New Zealand army draft of World War II. Access was given for this purpose to the Army Base Records, Wellington. To secure as perfect a sample as possible, names were drawn from files classified under each letter of the alphabet and in number proportional to the total number of entries under each letter. The names drawn from each letter-group (which followed a complete alphabetical sequence) were the ones, up to the number required, which came first. It would appear that in this way an almost perfectly random sample of the male population was secured. Height measurements of 21,342 and weight measurements of 21,177 men were thus available.

The analysis of the height measurements when arranged according to the months of birth follows in table 1 which gives the means in inches along with their probable errors and the number of cases. A more detailed analysis is presented in table 2, a frequency table giving a breakdown by months. The original calculations were based on half-inch intervals but for reasons of space inch intervals were used here. In following the months it must be remembered that the data are for the Southern Hemisphere and that the seasons are therefore reversd.

TABLE 1

Heights by Birth-Months

	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sep.	Oct.	Nov.	Dec.
Mean (inches)	68.19	68.25	68.09	68.06	68.10	67.94	68.16	68.18	68.24	68.16	68.24	68.17
P.E.*m*	.0409	.0415	.0392	.0389	.0406	.0390	.0392	.0414	.0391	.0406	.0423	.0400
number	1752	1634	1849	1763	1802	1799	1832	1720	1886	1861	1659	1785

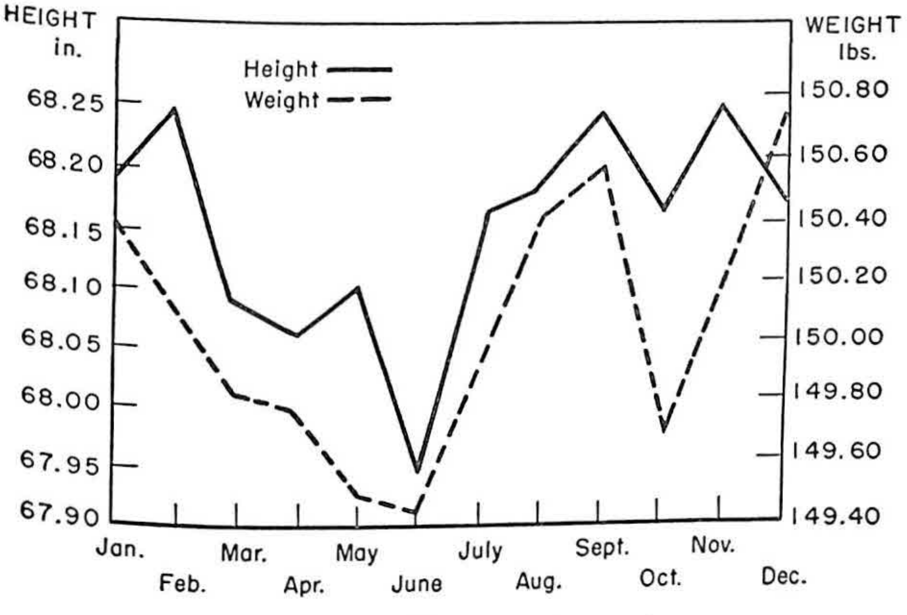

FIG. 1. HEIGHT AND WEIGHT BY BIRTH MONTHS.

Table 1 shows the tallest men were born in February and the shortest in June. Those of September and November birth are just shorter than those born in February. The differences between the June mean and those of these high-level months are significant. Some of the other high means when compared with the June low-level provide fairly significant differences. The mean difference between the tallest group and the shortest is just under a third of an inch (.31 inch). These monthly fluctuations are illustrated in figure 1. The main trends of this analysis correspond to those of studies reported by the author in 1941 with reference to the heights of children.

TABLE 2

Distribution of Heights by Birth-Months

Inches	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sep.	Oct.	Nov.	Dec.
50 Plus								1				
51 "								0				
52 "								0				
53 "								0				
54 "						1		0				
55 "						0		0				
56 "						0		1				1
57 "					1	0	1	0		1		0
58 "			1	2	1	0	2	0		0		0
59 "	1	1	0	0	1	0	2	2	2	4		0
60 "	4	5	4	4	4	4	6	2	4	5	2	1
61 "	11	4	5	6	8	2	5	5	8	6	5	12
62 "	27	20	21	18	20	35	20	16	23	29	20	17
63 "	38	43	64	46	54	56	45	36	46	53	45	50
64 "	92	68	93	96	111	113	98	98	87	105	83	99
65 "	152	143	182	171	158	156	139	161	161	153	139	178
66 "	218	218	229	259	230	273	251	245	250	236	206	219
67 "	251	263	315	262	281	262	306	248	294	277	254	244
68 "	306	233	266	265	276	307	293	267	297	296	272	289
69 "	250	226	251	251	229	226	257	246	244	236	225	253
70 "	171	190	191	178	184	169	181	151	215	193	172	186
71 "	109	108	117	99	130	100	112	116	118	127	103	110
72 "	59	69	62	60	68	57	64	73	75	81	53	78
73 "	43	24	28	33	25	23	26	32	41	42	47	32
74 "	12	15	12	10	14	12	12	13	15	10	25	13
75 "	4	4	6	3	3	0	8	6	2	5	7	2
76 "	3		1		3	2	3	1	4	2	1	1
77 "	0		1		0	0	1					
78 "	1				1	1						

The corresponding facts for weights according to month of birth are given in tables 3 and 4 and figure 1. The distributions in table 4 work

TABLE 3

Weights by Birth-Months

	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sep.	Oct.	Nov.	Dec.
Mean (lbs)	150.41	150.11	149.81	149.78	149.48	149.44	149.91	150.41	150.57	149.67	150.19	150.76
P.E.*m*	.3182	.3275	.3061	.3163	.3011	.3066	.3216	.3178	.2946	.3045	.3174	.3155
number	1758	1583	1822	1739	1799	1799	1825	1724	1849	1860	1638	1781

TABLE 4

Distribution of Weights by Birth-Months

Pounds	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sep.	Oct.	Nov.	Dec.
86 Plus						1		1				1
94 “	4	1		3	1	3	6	0	3	2	2	2
102 “	8	4	11	14	4	5	12	8	9	14	5	8
110 “	40	27	34	31	44	50	26	39	34	45	28	34
118 “	78	77	82	88	94	90	87	82	67	85	87	95
126 “	216	208	254	232	248	227	256	204	223	245	211	220
134 “	304	280	323	283	270	307	283	286	282	278	262	276
142 “	300	271	312	315	331	304	328	312	390	388	289	332
150 “	270	261	266	292	306	310	313	262	297	273	271	291
158 “	201	164	224	194	184	189	201	201	219	206	178	175
166 “	151	123	133	115	144	137	143	140	140	122	125	129
174 “	81	59	61	59	66	67	56	75	67	76	77	98
182 “	41	55	51	44	50	52	49	52	52	60	52	56
190 “	27	23	29	31	24	26	27	28	24	28	19	27
198 “	12	10	22	18	22	11	12	12	17	14	17	19
206 “	10	7	14	10	4	10	12	11	12	15	6	7
214 “	6	6	3	2	4	5	7	7	6	6	6	5
222 “	6	4	0	2	1	3	1	1	5	1	1	3
230 “	1	2	1	4	1	1	3	2	2	1	0	2
238 “	1	1	0	2	0	1	2	0		1	2	0
246 “	0		1		0		0	1				0
254 “	1		0		1		0					0
262 “			1				0					0
270 “							0					0
278 “							0					1
—							0					
—							0					
—							0					
—							0					
406 “							1					

with 8-pound intervals instead of the original 4-pound intervals. The lightest men are born in June and the heaviest those born in December with a difference of 1.32 pounds. The June measure is part of an extended low-level period and the December one similarly of a high-level period.

The differences between the lowest weight and the others are not highly significant. This is due partly at least to the skewing in the weight-distributions well indicated by table 4, a fact that is related to the tendency of the older men toward excessive weight. This feature in adult weight was noted by Kendall ('43).

SUMMARY

1. An analysis was made of the height and weight measurements of over 21,000 men of the New Zealand army draft for World War II.
2. The tallest men were those born in February (summer) and the shortest those born in June (winter). There was an average difference of about one-third of an inch between them.
3. The heaviest men were those of December birth and the lightest those born in June. There was an average difference of about one and one-third pounds between these weight-levels.
4. The main differences in height were significant and those in the weights had a lower level of significance.

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BOOK REVIEWS

Standard Values in Nutrition and Metabolism. Edited by ERRETT C. ALBRITTON. Pages xiii + 380, W. B. Saunders Co., Philadelphia, 1954. $6.50.

In view of the rapid, almost terrifying rate of accumulation of scientific data, largely quantitative in nature, a periodical critical sifting and compilation of reported results is an important supplement of research productive of new facts. Consequently, the idea of a comprehensive handbook of biological data has a real appeal.

This ambitious project, receiving substantial federal financial support, was entrusted to a special committee, as an organ of the American Institute of Biological Sciences, affiliated with the National Research Council. In addition to the volume on blood (published in 1952) and the present fascicle, it is planned to cover in separate volumes the fields of growth and reproduction, animal and plant physiology, biochemical composition, and toxicology. There is to be, also, an abridged handbook, covering all the areas.

The current volume consists of 233 pages of tables, 16 pages of diagrams, some 100 pages of bibliography indicating the source of the tabular entries, and an index. The readers of this journal are likely to be particularly interested in data on man's daily nutrient allowances (bringing out the unresolved differences between the American, Canadian and British figures); functions of chemical elements and vitamins, with signs of deficiency and excess; excretion of the end products of metabolism; and basal metabolism, including the presently proposed standards and a comparison with other norms. The standards commonly used heretofore in America were based on measurements made on subjects unaccustomed to the procedure and were higher, while the British standards were based on the lowest of repeated measurements on trained persons under rigorously basal conditions. As example, the earlier American (Boothby, Berkson, and Dunn, 1936), current American (Boothby, 1952), and the British (Robertson and Reid, 1952) standards for 25-year-old men are 40.3, 38.4 and 37.1 Cal/sqm/hr, respectively.

It is hoped that anthropometric data, important for the characterization of man's nutriture, will be included in one of the subsequent volumes.

JOSEF BROŽEK

Coronary Heart Disease in Young Adults. By MENARD M. GERTLER, PAUL D. WHITE, AND OTHERS. Pages xviii + 218, Harvard University Press, Cambridge, Massachusetts, 1954. $5.00.

Sanitation and the antibiotics are so successfully solving the health problems caused by other organisms that soon only the faults of the human organism itself may remain to plague us. That these, however, will be more than enough to engage all our research energy is obvious from a cursory glance at our vital statistics. In spite of great advances in diagnosis, surgery and medical care, it is obvious that there is as yet little progress towards the control and prevention of our self-generating ills that now dominate the health picture in this country.

From the standpoint of mortality, the outstanding problem is now coronary heart disease, which bids fair to account for half the deaths in the near future. The appalling frequency of coronary heart disease, however, is perhaps less significant than the fact that it attacks and kills some of us at an early age while some of us seem to be immune. Of course, we want to know why, but the first step towards answering this must be to discover who. In other words, how do the patients with coronary heart disease differ from their more fortunate fellows?

Fifteen years ago Dr. Paul D. White began to attack this question by comparing patients with clinically healthy controls, and the present volume is a report on a continuation of this inquiry with a hundred young (under 40) coronary patients and a suitable control series.

In many characteristics, including relative body weight, the coronary patients were not peculiar, but several significant differences were found. The coronary patients tended to be "stocky" or "mesomorphic," to have relatively high concentrations of cholesterol and of uric acid in the blood, and to produce saliva of rather special characteristics. These peculiarities are quite non-specific, but they are statistically significant.

Much has been made in the past of the fact that men are more prone than women to early coronary disease. In the present study, Gertler, White and their colleagues used various methods to explore the question of a masculinity factor, but the results are inconclusive. They found no difference between the patients and the controls in the excretion of male sex hormones. In physique, the patients tended to be more masculine than the controls but psychologically they were judged to be more feminine.

A dietary inquiry was also inconclusive except that it was clear that the coronary patients did not consume large amounts of cholesterol nor eat a very heavy diet at the time they were studied. However, this was the situation *after* the development of coronary disease and it would be more useful to know their lifelong dietary pattern *before* the development of the disease and all the resulting changes in the mode of life.

The work by Gertler, White and their colleagues was frankly exploratory. This clear presentation of their findings will help point the way to profitable new studies. By the same token their results tend to discount the probable value of further studies with some of the methods they tried. In sum, this book gives emphasis to the great need for larger and more systematic efforts to discover the characteristics of body and of mode of life associated with the development of atherosclerosis and coronary heart disease. It is probable that there is no single cause but only influences to be found and that all of us are susceptible in some degree. To discover the effective interplay of nature versus nurture will require attack from many approaches and with many methods. Future multidisciplinary research will build on these studies.

ANCEL KEYS

1951 Impairment Study. By the SOCIETY OF ACTUARIES. Pages 300, Society of Actuaries, New York, 1954. $7.50.

For some fifty years, the Society of Actuaries has been publishing in irregular intervals the results of medical impairment studies conducted upon material contributed by American insurance companies. The last major study concerned itself with a twenty-year period ending in 1928. Minor studies were conducted in 1936 and 1938. The present investigation was based upon the experience of 1935-1949 upon the lives of applicants who were medically examined and applied for ordinary insurance policies; 27 life insurance companies representing 70% of ordinary insurance in force in the United States and Canada agreed to take part in this investigation.

The purpose of the study was to make recent mortality experience available for analysis of the effects of specified "impairments" (such as overweight, family history, heart murmurs and other circulatory conditions) on death rates. The practical end was to provide a basis for adjustments in insurance premiums (extra premiums) to be paid by individuals with less favorable life expectancy.

As a control and a reference point, a basic mortality table was constructed reflecting the death rate for the corresponding period among those applicants who were considered unimpaired. Since in previous studies other basic mortality tables have been used, a comparison of the effects of a particular impairment in two or more periods is difficult. For example, if an impairment would show 150% of standard mortality currently and was rated the same in a previous study, it would not necessarily indicate that this impairment has produced comparable mortality in the two periods. It would merely mean that it produced 50% more mortality than the group which, in the corresponding period, was considered standard by the underwriters.

In interpreting the findings for purposes other than those for which they were originally designed, the limitations of the data must be considered. The statistics are derived from a selected sample drawn from among white male adults in the middle income group. It is not necessarily representative of the population as a whole. Mortality ratios were computed on the basis of the number of policies and not on the number of lives. That means that if a person held several policies, he appears statistically like several exposures, and if such person died, several deaths were recorded. While the control group was selected on the basis of a medical examination, the standards were probably not the same as a researcher would wish to apply in selecting a group of clinically healthy persons. The study did not concern itself with the uncovering of more basic associations but mainly with impairment classifications and indicators, some of which do not represent any specific clinical diagnosis but only reports of physical findings or laboratory analysis.

All of this does not mean that the study is not highly significant for the purposes for which it was conducted but that in its present form it is not particularly useful for the practicing physician or the medical investigator. This is not the fault of the authors, who have fulfilled their assignment rather well. It is the result of an unfortunate lack of agreement upon standards between the clinician, the researcher, and the interpreter of insurance medical findings, and of the practical limitations of medical examinations conducted in connection with the issuance of life insurance policies. In some instances, only very recent research work, conducted outside the framework of actuarial mortality investigations, has pointed to significant indicators of "impairment," in the sense of predictors of unfavorable health or longevity (*cf.*, smoking or, apparently, high fat intake and elevated level of blood cholesterol).

In the impairment studies of the Society of Actuaries, there is much material remaining dormant which is important to human biology. Additional criteria of impairment may usefully be added. The insurance industry has a unique opportunity to aid research because it is able to observe large groups of initially clinically healthy persons over long periods of time and evaluate the role of individual differences in "constitution" (with its anthropometric, biochemical, physiological, and psychological aspects) and in the mode of life.

E. H. KLEPETAR

America's Resources of Specialized Talent. The Report of the Commission on Human Resources and Advanced Training, Prepared by DAEL WOLFLE, Pages xviii + 332, Harper & Bros., New York, 1954, $4.00.

This book had its beginnings in the concern of the National Research Council, the American Council of Learned Societies, the American Council on Education, and the Social Science Research Council with the relation between the need for and the availability of highly trained specialists in many areas affecting national welfare and security. The report is the outgrowth of studies that began in 1949 with the appointment of the Commission, with Dael Wolfle as director of research. The work is premised on the assumption that this country's place in world affairs for years to come will depend upon effective utilization of its intellectual resources, and this leads directly into a series of fact-packed chapters that contribute answers, in some measure, to such questions as: What are the prospects for effective utilization of specialized talent? What are the future needs for it? What is the potential supply? How adequate are we utilizing these potentialities?

The report begins with a detailed classification of the fields of specialization. In general the Commission confines itself to those fields for which four or more years of college training are required. This leads into a comprehensive analysis of trends in college graduation by specialized fields, at both the bachelor's and the Ph. D. level. Another basic chapter is devoted to the occupational distribution of college graduates, with particular interest centering in the relationship between the field of college specialty and actual employment. This poses a fundamental question: To what extent is there flexibility in the utilization of trained manpower? The relationship between degree of specialization and ultimate use of trained ability has significant social implications.

The relation between supply and demand in the specialized fields is carefully analyzed, and as in all of the chapters, the assumptions that underlie the handling of the basic data are carefully stated. By general fields, present size of the professionally employed groups, estimates of current demand, and supply-demand prospects are set forth.

The logical question then follows: How much scientific talent is not being utilized; how much potential ability is being wasted? There is elaborate consideration of who goes to college and the ability-levels of the college population. The variations in intellectual ability among the specialized fields, by educational level, are striking evidence of a selective process that is going on.

Finally, the report discusses the utilization of the educated specialist and considers means for improving utilization of the potential supply.

Certain broad conclusions to which the Commission comes can be suggested here: the trend toward further specialization is unquestionable, and no evidence exists that a saturation point has been or is about to be reached. Because of the close relationship between college graduation and specialization, the future number of such graduates (now about 270,000; in 1970 to be about 590,000) is the key to future supply. Today, known shortages of specialists exist, and some degree of shortage can be expected to exist for many years, at least in some fields. While college graduates tend to remain in their field of original training, there is a large amount of shifting, and this flexibility is "a major national asset," since it contributes to more effective utilization of skilled manpower.

It has further to be concluded, the report demonstrates, that much potentially high talent is being wasted. Colleges could be double in size in this country with no lowering in quality. It is not that the college population is too big; rather, that a significant percentage of high ability youth do not go to college. Fields of specialization do select on an ability basis: students in physical sciences, business and home economics. These differences, overlapping as they are, are less marked than the differences between college and non-college groups.

The Commission believes there is waste in utilizing available trained ability and that there are steps that could be taken to improve this situation. There is need, too, for more attention to identifying, encouraging and even subsidizing the potentially able students so that they will receive the specialized training that existing and future needs call for.

No review as brief as this can do more than suggest the wealth of material assembled in this volume, and the ingenuity and care with which it has been drawn together and analyzed. Certainly the book does not answer all the questions that it raises, but it provides the best available starting point for the further investigations that should follow. It is a stimulating volume, not only because of the information it provides about existing human resources, field by field, but even more because of its broad implications that fundamentally touch national policy, and through policy, future national security.

MALCOLM M. WILLEY

Human Limbs and their Substitutes. Prepared under the sponsorship of the Advisory Committee on Artificial Limbs, National Research Council. Edited by PAUL E. KLOPSTEG and PHILIP D. WILSON, Pages 714, McGraw, Hill, New York, 1954, $12.00.

Three facts stand out as one attempts to assess the worth and significance of this large volume: 1) the range and diversity of the fields from which the contributors have been drawn; 2) the monumental nature of the work upon a subject which has previously had virtually no modern literature; and 3) the testimony it bears to the admirable coherence of a program which has been centralized about a pressing human problem but which requires for its solution the concerted action of diverse specialists.

The variety of contributing fields to the practice of prosthetics (limb fitting) is explained when it is realized that the stump is produced by the medical man, the artificial limb is fabricated by a paramedical specialist, the prosthetist, using mechanical components which are devised and manufactured by engineering and industry, and the amputee is aided in his biomechanical adjustment to the limb by the physical or occupational therapist, and in his emotional readjustment by the psychologist. Accordingly, there are chapters dealing with surgery, such as amputation, amputee pain and cineplasty; with prosthetics, such as prescription and limb fabrication; and with rehabilitation, such as training, therapy and psychological treatment.

Since it is also a report on recent research and development, the book contains much new and unique material in both the biological and technical categories. Exhaustive locomotion and manipulation studies are reported, and the outstanding developments in replacement mechanisms are described. These applications provide many significant biotechnical lessons. The excellent amputee pain studies represent advances in both theory and medical practice. The development of the cosmetic glove itself is a story of achievement in applied high polymer chemistry.

In summary, *Human Limbs* is an essential reference work for all those who work directly with amputees—surgeons, prosthetists, and therapists. But for many others it provides excellent source material on body mechanics, specialized engineering mechanisms and materials, and rehabilitation concepts and methods.

CRAIG L. TAYLOR

A Methodological, Psychiatric and Statistical Study of a Large Swedish Rural Population. By TAGE LARSSON and TORSTEN SJÖGREN. Acta Psychiatrica et Neurologica Scandinavica, Suppl. 89, Pages 250, Ejnar Munksgaard, Copenhagen, 1954, 25 Swedish krona.

This is the first volume of the newest of the Scandinavian population studies. It describes, for a change, not an isolate but a district selected as average and representative of Swedish rural population, thus providing a psychiatric base line for the whole of the country.

The ambition of the study, as seen in the first volume just published, has been the fullest feasibile ascertainment of the incidence of mental illness. The completeness of ascertainment is of utmost importance because the first step in genetic investigation of mental disorders is a comparison of familial concentration with incidence in the general population. This task has been completed satisfactorily; the study has furnished another set of reliable basic data, and this is its worth.

The authors have investigated the incidence of psychiatric disorders and of low grade mental deficiency in an area consisting of 13 parishes comprising two large and a number of small islands off the west coast of Sweden, close to the mainland. The area had a population of about 25,000 and there were some 1300 registered cases of major mental disorder. In order to avoid the well known but rarely heeded deficiencies of ascertainment inherent in the cross-sectional census method, the authors scrutinized their population through a 45-year period of observation. Their technique had been seasoned by previous Scandinavian field investigations. Most, if not all, of those investigations had been planned as a technical improvement on the population sampling methods initiated by the Munich school of psychiatric genetics. Judged from this viewpoint, this monograph represents a technical peak in the line of some four decades of endeavor to master the problems of sampling for the biological study of mental disorder.

The usability of their "period method" is largely limited to Sweden with her enviable system of registration. The selected period of observation, 1900-1944, covers the whole of development both of modern psychiatry and genetics. Certainly, the attitude toward mental illness was not the same at the turn of the century as it is at the present time. Clinical opinion, as well as diagnostic methods, too, underwent radical changes. Thus some uncertainty as to completeness and uniformity of ascertainment would be anticipated. The authors themselves acknowledge the shortcomings of this longitudinal observation by stating that the most representative figures were obtained from the period of 1920-1940. Aside from the uncertainties arising from the progress of time one may anticipate diagnostic imperfections inherent in any study depending for basic information on perusal of hospital records and field investigation. The authors are aware of that. One wonders if the labors and good will invested into such extensive field study could not be utilized in a more permanent manner, *e. g.*, by setting up a permanent clinic where a regular diagnostic job on a university hospital level could be done. Study of more neutral humain traits could be easily incorporated, and in this the human biologist would eagerly share.

The analysis of excess mortality linked to psychoses deserves attention. It is here calculated in terms of the remaining mean expectation of life. It has been again found that a psychosis which is not connected with frank somatic pathology shortens life significantly, although it does not kill directly. A few figures give a hint on biological forces which may make people move from their homes: about one quarter of the psychotics born in the area were not residing there at the time of the onset of their illness, which appears to be well in excess of the emigration rate. It can be inferred that certain aspects of the pre-morbid personality made easier the decision to migrate. This would tie in with Ödegaard's finding of a higher incidence of schizophrenia in Norwegian-born persons in Minnesota than in the population of Norway.

There is a great deal of thinking, experience and honest labor behind the many tables and the lengthy explanations of the procedure in the first volume of the monograph. The thoroughness of ascertainment of this study is in keeping with the tradition of Scandinavian population studies. The second volume probably will bring more in the way of general conclusions, and will be eagerly awaited.

KAREL PLANANSKY

BOOKS RECEIVED

HUMAN BODY SIZE AND CAPABILITIES IN THE DESIGN AND OPERATION OF VEHICULAR EQUIPMENT. By Ross A. McFarland, Albert Damon, Howard W. Stoudt, Alfred L. Moseley, Jack W. Dunlap, and William A. Hall. xiv + 239 pp. Harvard School of Public Health, Boston, 1953.

INDIAN HEALTH IN ARIZONA: SECOND ANNUAL REPORT OF THE BUREAU OF ETHNIC RESEARCH. By Bertram S. Kraus. ix + 164 pp. University of Arizona, Tucson. 1954. $1.50.

ATLAS OF MEN: A GUIDE AND HANDBOOK ON SOMATOTYPING. By William H. Sheldon. xvi + 357 pp. Harper & Brothers, New York, 1954. $10.00.

STATISTICS IN RESEARCH. By Bernard Ostle. xiv + 487 pp. Iowa State College Press, Ames, Iowa. 1954. $6.95.

THE PSYCHOLOGICAL VARIABLES IN HUMAN CANCER: A SYMPOSIUM. Edited by Joseph A. Gengerelli and Frank J. Kirkner. vi + 135 pp. University of California Press, Berkeley. 1954. $3.00.

HUMAN ENGINEERING GUIDE FOR EQUIPMENT DESIGNERS. By Wesley E. Woodson. 268 pp. University of California Press, Berkeley, 1954. $3.50.

EXPERIMENTAL PSYCHOLOGY: REVISED EDITION. By Robert S. Woodworth and Harold Schlosberg. xi + 948 pp. Henry Holt and Company, New York, 1954. $8.95.

GENETICA MEDICA: PRIMUM SYMPOSIUM INTERNATIONALE. Edited by Luigi Gedda. v + 467 pp. Istituto Gregorio Mendel, Rome. 1954.

ANALYSIS OF DEVELOPMENT. Edited by Benjamin H. Willier, Paul A. Weiss, and Viktor Hamburger. xii + 735 pp. W. B. Saunders Company, Philadelphia, 1955. $15.00.

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TASTE SENSITIVITY TO PHENYLTHIOUREA IN SPANIARDS

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INTRDUCTION

As is well known, phenylthiourea (P.T.C.) is a substance having a bitter taste. In 1932 Fox demonstrated notable individual differences in taste sensibility using this substance and many experiments using various methods have since been performed about the matter. Some authors use very simple techniques: for example, tasting of a simple aqueous solution or impregnated paper, or simply by putting pieces of crystal on the tongue.

Other investigators use more complex methods such as the use of a series of dilutions to determine the concentration threshold for each person. When this method is used it is seen that a simple classification into tasters and non-tasters is a reflection of an essentially bimodal distribution of taste thresholds and that the antimode can be taken as a ruler to differentiate the two categories. The dilution at which this antimodal minimum frequency occurs varies from population to population; methods which fail to take thresholds into account are subject to errors on this account. On the other hand, the subjective nature of the answers leads to errors which are difficult to control when the simpler methods are used, as well as, the latter one.

The method of Harris and Kalmus ('49), in which the subject is required to perform a sorting test, is an indubitable advancement for it permits a more objective determination of the threshold concentration. In this method we start with a stock solution of 0.13% and from it obtain a series of 14 solutions each with ½ the concentration of the previous solution. We used boiled tap water for these.

The test is performed in two stages. First, we determine the approximate value of the threshold by giving the subject a few cc of the solutions, beginning with the most dilute, until he perceives a definite taste. Secondly, we proceed to a more exact determination of the threshold by means of a sorting test. For this we give the subject 8 glasses, 4 having a few cc of water and the other 4 a few cc of the solution which has already been determined in the first stage of the test. The glasses are arranged at random and the subject is told that 4 of them contain the substance and 4 contain water; he is asked to separate them into the two groups. If he succeeds, we make him try with the next weaker solution and so on until he is unable to differentiate one of the solutions from water. If he is not able to establish the difference with the first solution, we do the same thing but follow an inverse order, using solutions of increasing concentration.

We believe that at present this Harris-Kalmus technique gives the most objective results. In the course of our studies we have verified that some suggestible individuals who, in the end, proved to be nontasters would have been classified as tasters had we followed their first subjective reactions and not done the sorting test. Furthermore, we have also tried the method of impregnated papers which are sold for this purpose and have found that the results are not always satisfactory. Apart from giving some wrong classifications, we sometimes got different answers for the same individual using different papers of the same origin—probably because the papers are not uniformly treated.

MATERIAL

The sample consists of 306 individuals: 203 males and 103 females. The majority are students or teachers at the University of Barcelona and the rest (13%) are otherwise employed. More than 85% of the individuals come from the northeastern provinces of Spain. Considering that the predominant racial element there is the Mediterranean, we venture to assert that this element is the most frequent one in our series. The sample is drawn from a large city, representative of the genotypic complex of the people of northeastern Spain; therefore, the sample does not correspond to a limited geographical breeding group.

It can be seen from the age distributions in tables 1 and 2 that the

TABLE 1

Taste thresholds for P. T. C. in Spaniards ♂

Age in years	Solutions															Total
	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	
6–7												1				1
8–9																
10–11																
12–13	1									1						2
14–15									1							1
16–17	4	2	1	1			2	2	6	8	9	5	1		1	42
18–19	2	6	4	3		1		4	11	21	8	3	1			64
20–21		2	1		1	1			2	3	6	1				17
22–23		2				1		1		4	3	2	1			14
24–25		2	2		1			1		4	3					13
26–27	2	1	2					1		2	1	2				11
28–29	2										2	1	1			6
30–31					1				1	1	1	3				7
32–33	1				1			1	2	4	2					11
34–35		1			1			1		1		1	1			6
36–37											2					2
38–39																
40–41		1														1
42–43			1													1
44–45									1							1
46–47											1					1
54–55										1						1
74–75										1						1
Total	12	17	11	4	5	3	2	11	24	51	38	19	5		1	203

ruler to differentiate the two categories. The dilution at which this antimodal minimum frequency occurs varies from population to population; methods which fail to take thresholds into account are subject to errors on this account. On the other hand, the subjective nature of the answers leads to errors which are difficult to control when the simpler methods are used, as well as, the latter one.

The method of Harris and Kalmus ('49), in which the subject is required to perform a sorting test, is an indubitable advancement for it permits a more objective determination of the threshold concentration. In this method we start with a stock solution of 0.13% and from it obtain a series of 14 solutions each with ½ the concentration of the previous solution. We used boiled tap water for these.

The test is performed in two stages. First, we determine the approximate value of the threshold by giving the subject a few cc of the solutions, beginning with the most dilute, until he perceives a definite taste. Secondly, we proceed to a more exact determination of the threshold by means of a sorting test. For this we give the subject 8 glasses, 4 having a few cc of water and the other 4 a few cc of the solution which has already been determined in the first stage of the test. The glasses are arranged at random and the subject is told that 4 of them contain the substance and 4 contain water; he is asked to separate them into the two groups. If he succeeds, we make him try with the next weaker solution and so on until he is unable to differentiate one of the solutions from water. If he is not able to establish the difference with the first solution, we do the same thing but follow an inverse order, using solutions of increasing concentration.

We believe that at present this Harris-Kalmus technique gives the most objective results. In the course of our studies we have verified that some suggestible individuals who, in the end, proved to be non-tasters would have been classified as tasters had we followed their first subjective reactions and not done the sorting test. Furthermore, we have also tried the method of impregnated papers which are sold for this purpose and have found that the results are not always satisfactory. Apart from giving some wrong classifications, we sometimes got different answers for the same individual using different papers of the same origin—probably because the papers are not uniformly treated.

MATERIAL

The sample consists of 306 individuals: 203 males and 103 females. The majority are students or teachers at the University of Barcelona and the rest (13%) are otherwise employed. More than 85% of the individuals come from the northeastern provinces of Spain. Considering that the predominant racial element there is the Mediterranean, we venture to assert that this element is the most frequent one in our series. The sample is drawn from a large city, representative of the genotypic complex of the people of northeastern Spain; therefore, the sample does not correspond to a limited geographical breeding group.

It can be seen from the age distributions in tables 1 and 2 that the

TABLE 1

Taste thresholds for P.T.C. in Spaniards ♂

Age in years	Solutions 0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	Total
6–7												1				1
8–9																
10–11																
12–13	1									1						2
14–15									1							1
16–17	4	2	1	1			2	2	6	8	9	5	1		1	42
18–19	2	6	4	3		1		4	11	21	8	3	1			64
20–21		2	1		1	1			2	3	6	1				17
22–23		2				1		1		4	3	2	1			14
24–25		2	2		1			1		4	3					13
26–27	2	1	2					1		2	1	2				11
28–29	2										2	1	1			6
30–31					1				1	1	1	3				7
32–33	1				1			1	2	4	2					11
34–35		1			1			1		1		1	1			6
36–37											2					2
38–39																
40–41		1														1
42–43			1													1
44–45									1							1
46–47											1					1
54–55										1						1
74–75										1						1
Total	12	17	11	4	5	3	2	11	24	51	38	19	5		1	203

majority of subjects were fairly evenly distributed within the age range 16–37 years. The mean age is 22.3 years for the males and 24.0 years for the females. The threshold values in these tables were determined exactly as indicated by Harris and Kalmus.

TABLE 2

Taste thresholds for P. T. C. in Spaniards ♀

Age in years	Solutions 0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	Total
10–11											1					1
12–13																
14–15											1					1
16–17		3							1	2	3	1				10
18–19		4		1	1			2	1	7	4	2	3			25
20–21		1	1					1	1	2	4	5				15
22–23	2		1	1					1	5	3	1		3		17
24–25									1	1	1		1			4
26–27		1								1	1					3
28–29	3		1		1						1	1	2		1	10
30–31											3					3
32–33		1								1						2
34–35										1	1					2
36–37										1						1
38–39											1		1			2
40–41			1								1	1				3
42–43										1						1
44–45																
46–47															1	1
54–55	1															1
64–65										1						1
Total	6	10	4	2	2			3	5	23	25	11	7	3	2	103

RESULTS

Sensibility thresholds

Sexual dimorphism. Considering that the mean age of both series and the distribution of frequencies at different ages are similar, the masculine series can be compared directly with the feminine one. The average taste threshold for males is 7.33 and for females, 7.98. That is, the average taste sensitivity is greater in women than in men which accords with results given by other investigators working on other populations.

Apart from the above considerations, the two distributions (see figure 1) were compared using the χ^2 test; the difference is slightly high but not statistically significant ($\chi^2 = 16.59$; probability differential laying between 90% and 95% for 10 degrees of freedom). Classes 4, 5, 6 and 12, 13, 14 were merged for the χ^2 calculation.

Influence of age. Harris and Kalmus ('49) in their studies on English people and Mohr ('51-'52) on Danish, found that taste capacity to phenylthiourea decreases with age. To study this we have combined the masculine and feminine series; as noted above, the age distribution in the two sexes is alike.

Our series is chiefly comprised of first year university students. For this reason we can only give few data on older individuals. The results are as follows:

Age	n	Mean threshold
x–19	147	7.44
20–39	146	7.69
40–x	13	7.23

From these we can see that taste capacity does not decrease up to the age of 40. From that age on, the small number of data does not permit any conclusion.

Influence of tobacco. It has been said that use of tobacco might decrease the capacity to taste phenylthiourea. To check this in Spaniards we questioned the individuals here analysed concerning their use of tobacco. If they answered affirmatively we asked them the average number of cigarettes they smoked daily. As women in Spain are generally non-smokers, we dispensed with the feminine series and based this study on the masculine series.

There were 97 male non-smokers and 75 individuals who smoked at least 5 cigarettes daily. The average threshold of the former is 7.38 and of the latter, 7.39. This verifies, by a different technique, the conclusion of Falconer ('47) that tobacco does not seem to influence this taste capacity. Furthermore, a subseries of 52 individuals who smoked 10 or more cigarettes daily was elaborated for greater sureness. The taste threshold average is 7.52, that is, even slightly higher than non-smokers.

Frequency of non-tasters

As there is no apparent variation of taste sensibility with age in the ages covered in this series, one can dispense with the subdivision into age groups and use the total series for the determination of the antimode.

For the males it seems convenient to separate the tasters from non-tasters between classes 5 and 6. For the females there is no problem; the distribution is discontinuous and there are no cases in classes 5 and 6. Futhermore, for both series combined, the division at a limit between classes 5 and 6 seems still most reasonable. The same would be true if an antimode were established for each age group separately.

Dividing the series at this limit we obtained the following frequencies:

	Non-tasters	Tasters	Total	% non-tasters		Standard error
Male	52	151	203	25.6	±	3.1
Female	24	79	103	23.3	±	4.2
Total	76	230	306	24.8	±	2.5

The frequency of non-tasters is greater in the males, but the difference is not statistically significant ($\chi^2 = 0.196$ for one degree of freedom).

The proportion of non-tasters was also calculated according to smoking habits. The differences are very small: non-smokers, 24.7%; smokers (at least 5 cigarettes daily), 25.3%; and heavy smokers (10 or more cigarettes daily), 25.0%.

We have also attempted a comparison with other human groups. In the pertinent literature there are considerable data on the proportion of tasters and non-tasters in different populations. Much of this was collected by other techniques, however, and, as we have already indicated, comparisons between populations may not be meaningful under such circumstances. For this reason we shall here limit comparisons to data obtained by the Harris and Kalmus technique.

In table 3 we give data for different populations following an increasing order of non-tasters. This permits one easily to see the contrast between the European populations proper, with frequencies of non-tasters of 25% to 32%, and African Negroes and Chinese and also the Lapps, with much lower frequencies.

TABLE 3

Frequencies of non-tasters in different populations using serial dilutions with sorting test

Population	Number	% non-tasters	Authors
African Negroes	74 (71 ♂ + 3 ♀)	2.7	Barnicot ('50)
Norwegian Lapps	78	6.8	Allison and Nevanlinna ('52-'53)
Swedish Lapps	62	7.0	Allison and Nevanlinna ('52-'53)
Chinese	66 (55 ♂ + 11 ♀)	10.6	Barnicot ('50)
Spaniards	306 (203 ♂ + 103 ♀)	24.8	Pons (The present study)
Finns	202	29.2	Allison and Nevanlinna ('52-'53)
English	441 ♂	31.5	Harris and Kalmus ('49)
Danes	314 (194 ♂ + 120 ♀)	31.8	Mohr ('51-'52)

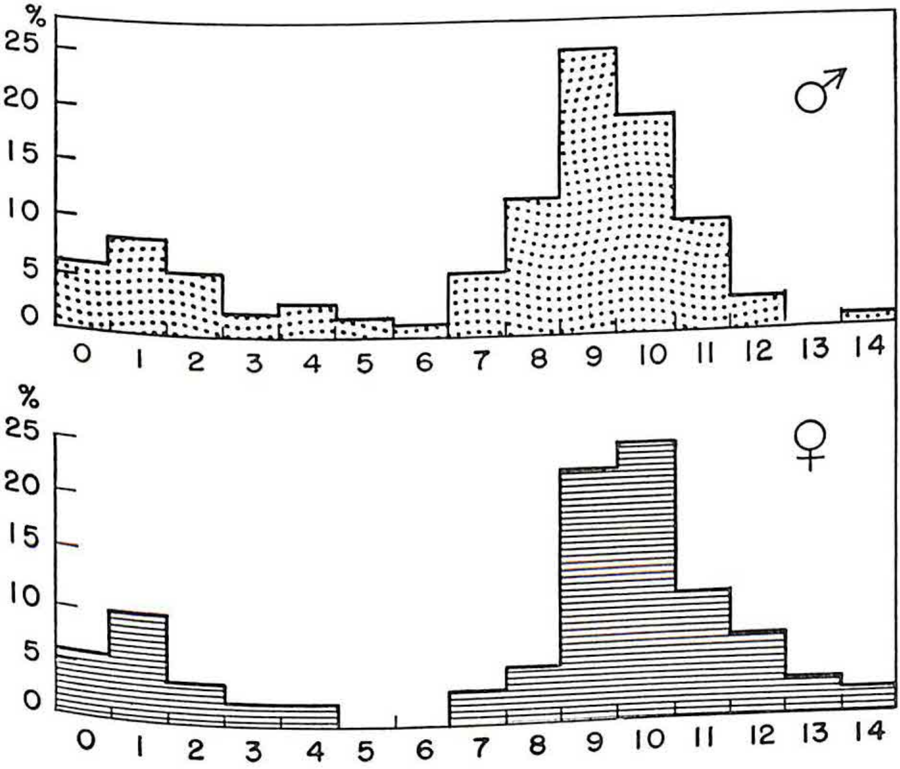

FIG. 1. DISTRIBUTION OF TASTE THRESHOLDS IN SPANIARDS.

SUMMARY

Taste sensitivity to phenylthiourea was investigated in 306 Spaniards by the Harris and Kalmus technique. The average threshold is somewhat higher in females than in males. This is in agreement with earlier observations. Smoking does not reduce the capacity to taste phenylthiourea.

The frequency of non-tasters in Spaniards is 24.8%. A comparison of the present data with other populations so far investigated by the same technique shows that Europeans have high frequencies of non-tasters when compared with other populations.

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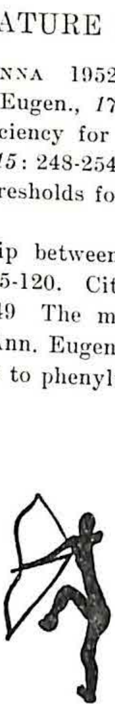

DEPOPULATION ON ULITHI

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INTRODUCTION

ULITHI Atoll, in common with most of the islands of the Caroline Archipelago, has experienced a severe decline in population since the early part of the century. It is my purpose to set down here some facts and figures to support certain opinions which I hold regarding the causes of this decline. Any contribution I may make will have to be a modest and tentative one, for the vital statistics and medical data necessary to construct an airtight case are not available. Nevertheless during a total of 9 months which I spent on Ulithi, distributed in two trips covering 1947-49, I made enough observations and collected enough census materials to convince me that my conclusions have much validity.

The atoll is located at 10° north of the equator at 139° east longitude. It is about 400 nautical miles southwest of Guam. The islands are composed of a coralline soil which supports only a limited variety of plant crops, particularly the coconut, taro, squash, sweet potato, and breadfruit. Fish contribute virtually the only item of flesh food. Social organization is based on matrilineal clans and lineages with patrilocal residence. There is almost no social stratification. Political control rests mostly in the hands of village councils which exercise their authority with wisdom and justice. Constant contacts of political and economic character are maintained with nearby islands, especially Yap, Ngulu, Fais, and Woleai. Most of the people are now Roman Catholics, having been converted since 1937, but there are many older people who still subscribe to a religion based on animistic beliefs, mild ancestor worship, and otiose deities. There is a strong belief in magic, both black and white. The people seem to be psychologically well adjusted, optimistic, and concrete, with an overwhelmingly group-oriented approach (Lessa and Spiegelman, '54).

There is a faint possibility that Ulithi was first discovered in 1525 by the Portuguese captain, Diego da Rocha. The evidence for this has with justice been disputed. Similarly doubtful is the often repeated assertion that the atoll was discovered in 1528 by the Spaniard, Alvaro de Saavedra, and that he remained there about a week. It is much more probable that the first to sight the island group was Ruy Gomez de Villalobos on January 23, 1543. While there is some argument that he may have seen Yap instead, there is no doubt that he visited some of the islands in the vicinity of Ulithi. Thus, it was probably the island of Fais, less than 40 miles to the east, where he found some natives who sailed out in boats and made the sign of the cross with their hands, saying in Spanish, "Good day, sailors!" This indicates that there had been a prior visitor in the area. Perhaps it was the aforementioned Diego da Rocha, for he seems to have been the first to discover Yap, on which he stayed for a while. But none of these voyages could have had an appreciable effect on the population of Ulithi, for either there was no more than fleeting contact or no contact at all. Moreover, the period involved was one in which venereal diseases had not gained great headway among Europeans.

There could have been more effect from "seven white men who wore pantaloons and stockings" and remained on the atoll, 5 of them marrying native women. These men were described by Olit, a boy who was one of a party of shipwrecked natives from Fais who landed in the Philippines in 1696 after 70 days at sea. There is no reason to suppose, however, that these settlers had anything to do with introducing factors making for depopulation. Other contacts, too, coming soon after, seem to have had little permanent effect. For example, the next visitor to Ulithi was Don Bernardo de Egui y Zabalaga, who arrived in 1712 and was involved in a disagreeable incident involving the wounding of some natives by musket fire. Egui had little time other than to antagonize the natives, whom he tells us were a "well-built race" and "very gay." Judging from the spirited opposition they put up when he attempted to abduct one of their number, they were not a demoralized people.

The next visitor to the islands stayed long enough to have left an imprint but it is doubtful if he or his party had any effect on population. Father Antonio Cantova had had a long and ardent desire to convert the natives, about whom he had learned in 1721 from some Carolinians shipwrecked on Guam. He ultimately arrived on Ulithi on March 2, 1731, accompanied by another priest, 12 soldiers, 8 cabin boys, and a Carolinian. After three months the party ran out of food and the other priest was sent back to the Marianas for help. He was delayed by storms and did not return until 1733, when he found all of the group of the mission had been killed. From that time on Ulithi was generally avoided because of the reputation it had acquired through the massacre.

Contacts became accelerated in the last century. The Russian explorer Lütke paid a brief visit in 1828, and the French explorer Dumont d'Urville passed through in 1838. In the latter half of the century the rival governments of Spain and Germany began to show activity in the area and contacts with the outside world began to be more frequent. After the Germans acquired formal control just before the turn of the century, Ulithians began to be subjected to their first real contacts with civilization. Many of the men went to work for the Germans, usually as miners on Angaur and elsewhere. When the Japanese took over in 1914 they exerted an even greater influence, especially by setting up a school system on nearby Yap. While the culture of Ulithi has remained surprisingly intact in the face of these influences, there can be no doubt that diseases from the outside world now for the first time became real problems.

POPULATION STATISTICS

Published vital records for Ulithi, insofar as the writer knows, are virtually nonexistent, so that any discussion of population will have to be based on a rough approximation. The one exception to this unfortunate situation is a precise census taken by the writer as of January 1, 1949.

An explanation of the method of conducting the census is useful. The actual gathering of the data was begun on July 19, 1948, and continued intermittently until January 20, 1949. A mimeographed form was used which allowed for the recording of the following: name; sex; age; marital status; adoption; residence; birthplace; lineage or clan; name, birthplace, lineage or clan, and state of existence of father, mother, and spouses; the number of children by each marriage; the cause of termination of each marriage (divorce, death); religious belief (pagan, Catholic); extent of travel beyond Ulithi; nature of foreign contacts; linguistic aptitudes; outstanding pathologies; miscellaneous. The start of the census was made on Mogmog, where reliable informants named all persons known to them by memory and supplied preliminary data, such as sex, approximate age, and residence. This information was checked with separate lists sent in from outlying islands by designated agents. For several months the data on each personal census sheet were expanded through material derived from genealogies, information provided by selected informants fetched from other islands, direct interviewing of each individual inhabitant wherever feasible, and the recording of current changes in the vital statistics (births, deaths, marriages, divorces). A final check was made, just before and after January 1st, by gathering together on each island all the available inhabitants. In their presence and with their aid the census forms were put in order of birth from youngest to oldest. Ages were then recorded, in some cases being exact because dates of birth were known, and in other cases being approximate, in which event a system of interpolation with the known dates as guideposts was used. Here it was helpful, too, to make reference to historical incidents (typhoons, onset of the wars of foreigners) for estimating the ages of individuals not close in age to other individuals whose dates of birth had been well approximated. Visual inspection was used only as a crude adjunct. Excluded from the census were all transient residents; included were any Ulithians briefly absent on other islands.

This census (table 1 and figure 1) revealed that there were 200 males and 221 females on Ulithi, making a total of 421 permanent inhabitants. The population of each island, from largest to smallest, was: Mogmog, 142; Falalop, 126; Fassarai, 69; Asor, 53; and Lossau, 31. No other islands are at present inhabited, although we know that there were people living on Sorlen, Pigelelel, Mangejang, and Lam in the early part of the century.

It is evident from the population pyramid that there are few children and a high proportion of older people. In the absence of significant net migration this indicates low fertility and high longevity. The reasons for the rather large number of individuals in the 5–9 group are being investigated.

Comparative population figures for Ulithi are meager and not wholly reliable but they doubtless indicate that there has been a severe decline in population since early in the century (table 2). In 1904 Senfft ('04) made what seems to be a reasonably accurate estimate. From that year to 1949 the total declined to approximately 53% of what it had been at that time.

The 1904 figures can be broken down according to island (table 3),

TABLE 1

Population according to residence, age, and sex (1949)

Age	Mogmog			Asor			Falalop			Fassarai			Lossau			Total		
	♂	♀	T	♂	♀	T	♂	♀	T	♂	♀	T	♂	♀	T	♂	♀	T
0	0	1	1	2	1	3	2	1	3	0	1	1	0	1	1	4	5	9
1	1	0	1	0	0	0	1	0	1	0	0	0	0	0	0	2	0	2
2	2	0	2	0	1	1	0	1	1	1	1	2	0	0	0	3	3	6
3	0	0	0	1	2	3	1	0	1	0	0	0	0	0	0	2	2	4
4	0	0	0	0	2	2	0	0	0	2	1	3	0	0	0	2	3	5
0–4	3	1	4	3	6	9	4	2	6	3	3	6	0	1	1	13	13	26
5–9	14	13	27	1	3	4	4	1	5	1	2	3	0	4	4	20	23	43
10–14	2	5	7	0	0	0	2	1	3	3	1	4	3	2	5	10	9	19
15–19	8	4	12	2	5	7	1	3	4	2	2	4	0	0	0	13	14	27
20–24	6	7	13	3	1	4	4	3	7	2	1	3	0	0	0	15	12	27
25–29	5	4	9	3	0	3	3	5	8	4	3	7	0	0	0	15	12	27
30–34	2	1	3	2	1	3	3	6	9	1	1	2	1	2	3	9	11	20
35–39	3	9	12	2	3	5	2	0	2	2	2	4	1	0	1	10	14	24
40–44	3	5	8	1	1	2	6	10	16	5	0	5	0	1	1	15	17	32
45–49	6	3	9	0	0	0	7	8	16	1	0	1	2	1	3	16	12	28
50–54	6	5	11	0	1	1	3	2	5	3	6	9	1	2	3	13	16	29
55–59	3	3	6	1	3	4	5	7	11	0	4	4	0	0	0	9	17	26
60–64	5	3	8	2	5	7	5	2	7	1	1	2	1	2	3	14	13	27
65–69	2	0	2	1	1	2	4	1	5	3	4	7	2	1	3	12	7	19
70–74	2	6	8	0	1	1	4	6	10	3	1	4	0	1	1	9	15	24
75–79	0	1	1	0	0	0	3	6	9	1	2	3	0	0	0	4	9	13
80–84	1	1	2	0	1	1	0	2	2	0	0	0	0	1	1	1	5	6
85–89	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0	1	1
90–94	0	0	0	0	0	0	1	0	1	0	0	0	1	1	2	2	1	3
Total	71	71	142	21	32	53	61	65	126	35	34	69	12	19	31	200	221	421

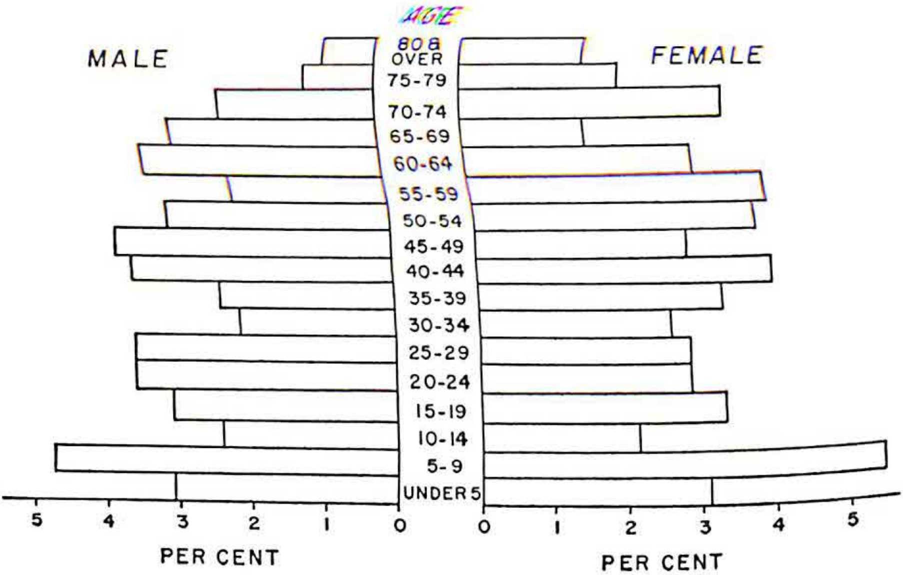

FIG. 1. AGE-SEX POPULATION PYRAMID FOR ULITHI (1949).

TABLE 2

Total population in various years

YEAR	POPULATION	SOURCE OF DATA
1870	ca. 700	Tetens & Kubary (1873)
1903	797	Senfft ('04)
1914?	ca. 700	Finsch ('14)
1925	506	Hasebe ('28)
1930	450	Hydrographic Office ('38)
1935	443	Hydrographic Officer ('45)
1946	402	U. S. Navy ('48)
1949	421	Lessa (present study)

indicating that one fourth the decline is due to the absence of people on the islands of Pigelelel, Sorlen, Mangejang, and Lam. Some of these people undoubtedly merely moved to other islands, e.g., Mogmog, where there has actually been an increase in population. But the figures for all the other islands now inhabited show strong losses, especially on Falalop, the largest of all the islands in the group, and Asor.

TABLE 3

Number of inhabitants on each island of Ulithi in 1904 and 1949

ISLAND	POPULATION 1904 (Senfft)	 1949 (Lessa)
Falalop	260	126
Asor	101	53
Mogmog	95	142
Fassarai	79	69
Pigelelel	79	—
Sorlen	70	—
Lossau	59	31
Mangejang	42	—
Lam	12	—
Total	797	421

Part of the severe drop between 1904 and 1925 can be attributed to the great typhoon of March 30, 1907, which caused great damage. Of this disaster we have the following account from German records: The storm came in the morning from the west, overthrew the palms and other trees, and destroyed the houses. But there was no flood tide. About noon the storm veered towards the west, and in the afternoon it struck the island again, and finally that evening it subsided. The canoes came from all the other islands to Tharex, and begged him to pray to Yonelap to protect them. Tharex cast a spell, and then it began to rain. For a time the inhabitants had to eke out an existence with fallen nuts and pumpkins [squash], which are cultivated in great quantities. The Schooner "Germania" took the natives who wanted to leave to Yap and Saipan, so there was no famine (Damm, '38. *Cf.* also Kurtz, '07.).

The present inhabitants of Ulithi are inarticulate regarding this typhoon and its effects. Since repeated questioning has elicited little information of value, we are forced to rely on other sources. We know that whereas in 1904 there were 260 persons on the island of Falalop, there were only a little over a fourth that number in 1909, two years after the typhoon. This was the year when Ulithi was visited by members of the Thilenius expedition. The members of the expedition reported that according to Hellwig's and Lorenzen's estimations the population of Falalop consisted of about 100 persons; both had based their estimates on the number of dwelling houses. Hellwig, however, discovered a number of buildings that stood at present deserted or whose dwellers had died, so that 70 persons would be a more accurate estimate; but those inhabitants who might return from Yap, Angaur, and Saipan must also be taken into account (Thilenius and Hellwig, '27).

The question immediately arises as to what happened to the people as a result of the typhoon. Some departed from Ulithi for other islands, *e. g.*, Yap, Angaur, and Saipan. From the census data gathered by the present writer, we know that a good many people remained on these other islands for a long time and there gave birth to many individuals who now reside in Ulithi. Thus, a woman by the name of Lemaremokh was born on Saipan ca. 1917 and returned to Ulithi ca. 1928. Four others were born there, ca. 1912, 1915, 1918, and 1920, respectively, so their parents were on Saipan during those years. The writer has no data on whether the movement back to Ulithi was in dribbles or occurred at one time, but, according to informants, there are still many Ulithians living on Saipan. As for the other islands to which the Germans transported the refugees, we know that three Ulithians who are now adult were born on Yap ca. 1913, 1916, and 1918, respectively, their parents having presumably gone there after the typhoon. One Ulithian was born in 1913 on Angaur, of parents who probably had sought haven there.

Further verification of the effects of the typhoon can be seen in figure 2, which shows that the number of persons born before the storm was considerably greater than after it. That is, of those Ulithians now living more were born per year immediately before the typhoon than immediately after it. The average for 1903-1907 was 7.4 per year, whereas the average for the post-storm years of 1908-1914 was 4.0 per year. The downward character of the curve gains greater significance when it is kept in mind that attrition through age has been greater for those born in the earlier years than those born in the later ones. Admittedly, this method has its limitations, but I devised it as a substitute for actual birth records for the years involved. Some other reason may account for the drop since 1907, but I do not know what it may be.

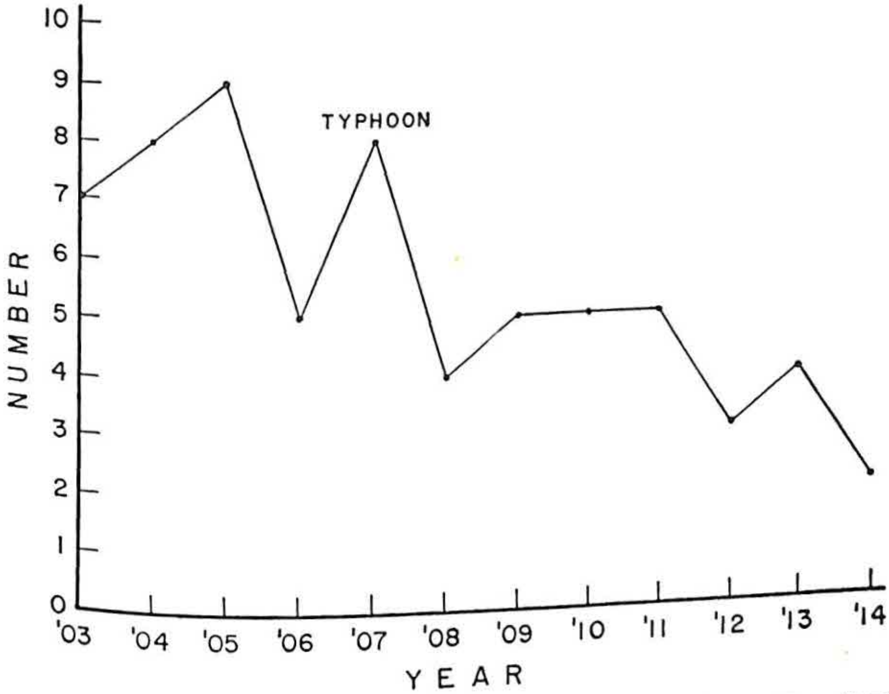

FIG. 2. NUMBER OF INDIVIDUALS LIVING ON ULITHI (1949) WHO WERE BORN BEFORE AND AFTER THE TYPHOON OF 1907, BY YEAR OF BIRTH.

A CONSIDERATION OF CERTAIN DEPOPULATION THEORIES

Having shown that there indubitably has been a decline in the population of Ulithi it is now in order to inquire into its causes. Since the typhoon of 1907 has held so important a place in the previous discussion we shall first evaluate it and other typhoons in general. Guided by the principle that a phenomenon which is not new can hardly explain a new trend, I have rejected the 1907 storm and all others as accounting for the present paucity of population. There are several reasons for this. Apparently no one was killed by the wind storm, and the German government provided early and sufficient aid. The immediate drop in population resulted from the removal of many natives to islands not struck by the typhoon, but most of these people eventually returned to their homes. If the storm had been truly castastrophic it should have left a greater impress on living residents, yet they know little about it. When Hellwig in 1909 asked the widow Suliven why Falalop had been so depleted of people she gave as her three reasons: an epidemic, the hiring of men to work abroad, and the loss of traveling canoes on the high seas (Thilenius and Hellwig, '27). It is notable that she did not list the typhoon as a factor. Certainly it seems to have nothing whatsoever to do with the small size of present day families, for births in the more recent years prior to 1949 have been abnormally low, without any indication of an upward movement in the population curve that would help recover the losses due to the typhoon. After all, this is a typhoon area and undoubtedly there have been many storms as severe as the one in 1907, which merely happens to have been fairly well recorded by the Germans. With the advent of colonial government with its modern facilities, the net effect of most storms seems to be that it results in the shifting about of peoples from one area to another, usually temporarily. It is interesting that many people now living, however, as permanent residents on Ulithi arrived there as refugees from typhoons on their own islands. In a case such as this, then, typhoons may create an increase in population through immigration.

Not much stock should be placed, either, in those theories of Micronesian depopulation which claim the loss of men at sea as a major contributing factor. Of course, the hazards of sea voyages in native proas are considerable in this far flung, typhoon battered area. The widow mentioned above was obviously impressed by the number of men who had lost their lives at sea in this manner. The songs still chanted on Ulithi often tell of the death of dear ones traveling to distant lands. The literature, from as far back as the 17th century, speaks constantly of the Europeans who have given aid to men blown from their courses. Earlier we saw how in 1721 some shipwrecked Ulithians came to the attention of Father Cantova on Guam. Parkinson writes of some natives of Ulithi who, in 1903, were being repatriated after drifting 900 nautical miles and landing on Halmahera. Senfft reports that in 1898 a typhoon struck a fleet of Ulithian canoes traveling to Fais and 120 people disappeared, and that later 7 canoes went out to search for these people but instead were blown to the Philippines. The Germans became so vexed at the expense they were incurring in the repatriation of survivors that they did their best to prevent travel in native canoes. As for the Japanese, they were not long in forbidding it by law.

Yet, while there is no doubt that many Ulithians have lost their lives at sea, this is not a serious cause of depopulation. The shipwreck theory has this weakness: it does not take into consideration that loss of life at sea is a constant over a long period. Indeed, there is every evidence that such loss of life was more common prior to depopulation than after. The shipwreck theory would hold good only if it could be demonstrated that travel on the high seas had increased, or that the art of navigation had declined, or that typhoons had increased in frequency. We know that the Micronesians were able seafarers, approaching the great Polynesian tradition. Another argument against the theory is that, whereas men may lose their lives at sea, the women they have left behind will continue to bear children, for it is an established fact that adultery and premarital license are common practices in the Carolines, as they must have been throughout all Micronesia. The shipwreck theory does not account for the fact that Fais, whose natives are known as poor navigators who seldom take to the high seas, have declined even more rapidly than Ulithians in number, despite a sex ratio of 127, according to data gathered in 1905 by Senfft ('06).

Nor does the author believe, as far as Ulithi is concerned, that depopulation has resulted from abortions, infanticide, or the use of contraceptives. The fact of the matter is that there is no attempt to curtail births. I could find no evidence of birth limiting practices. Ulithians want dearly to have children. Besides, any theory of depopulation which attributes decline to such factors would have to show that they had increased from times past when there had been a more abundant population.

Infrequency of coitus, which has been advanced as a cause of decline in Yap (Hunt *et al.*, '54), is another theory which cannot be given credence for Ulithi. While the author has not collected specific data on the topic, it seems obvious from having lived among the people for 9 months that intercourse is a pleasurable activity indulged in with great frequency. Adultery is common and premarital relations are readily permitted. It is true that for long periods women are confined to menstrual huts, so that men are not accessible to them. It is also true that abstinence is required by certain taboos. But these factors are not new ones; they have existed from the remote past. If anything, there are fewer occasions on which, for religious and other reasons, continence is required. The decline of paganism has meant a relaxing of the rules.

Inbreeding is not a cause of depopulation on Ulithi, since actually the atoll is not as endogamous as one might suppose. For one thing, the incest range is so wide that it forces people to select mates not allied by anything but the remotest ties. For another, census figures show that despite the great distances which separate the islands of the Carolines, there has been much migration. While some of this movement is recent and has been fostered by administrative authorities, most of it is aboriginally inspired. For Ulithi, this is verified by an examination of genealogies, which shows ancestors coming from 9 islands outside the atoll. In 1909, according to Krämer, there were about 20 Fais women living on Ulithi. Table 4, which limits itself to the parents of the permanent residents of the atoll, provides exact evidence of the mixed character of the population. It is based on census figures for 1949. The high percentage of persons from Sorol is due to an influx following a typhoon. The large number of persons born on Fais reflects the closeness of that island to Ulithi. The relatively low percentage of people from nearby Yap is undoubtedly a result of caste restrictions. Some of the most important lineages and clans of Ulithi are, according to tradition, from distant islands in the Carolines. In view of the fact, then, that at least 15% of the people of Ulithi have "foreign" blood, so to speak, we can consider that a salutary exogamy obtains.

TABLE 4

Provenance of the parents of Ulithian residents (1949)

PLACE OF BIRTH	PERCENTAGE
Ulithi	80.0
Fais	4.4
Sorol	4.4
Woleai	1.8
Saipan	1.3
Ifalik	0.7
Yap	0.7
Lamotrek	0.6
Angaur	0.4
Ngulu	0.2
Faraulep	0.1
Eauripik	0.1
Unknown	*5.3*

Ulithians remarry often, mostly because of divorce. There is no polygamy, for the marriages are always successive. Table 5 shows the number of times, by age groups, that individuals have been married. Younger persons, of course, have had less time to remarry, whereas older ones have had more opportunity. Therefore, while the average number of marriages contracted by all persons who have married is 2.05, that for persons 50 years of age or over is 2.46. Men marry more often than do women, the average for persons 50 years of age and over being 2.62 spouses for males and 2.34 spouses for females. These figures are conclusive evidence that marriage is a "brittle" affair on Ulithi, and if the memories of the older natives were more accurate on this score, it would be found that the average number of marriages contracted during their lifetimes would be even higher. The truth of the matter is that some Ulithians fail to recall, unless prompted, certain of the

TABLE 5

Number of marriages per individual by age groups (1949)

AGE GROUP	NUMBER OF MARRIAGES											
	Males						Females					
	1	2	3	4	5	6	1	2	3	4	5	6
10–14	0	0	0	0	0	0	2	0	0	0	0	0
15–19	1	0	0	0	0	0	12	1	0	0	0	0
20–24	7	2	0	0	0	0	7	4	0	0	0	0
25–29	8	6	0	0	0	0	4	7	0	0	0	0
30–34	5	2	0	0	0	0	5	2	3	1	0	0
35–39	6	4	0	0	0	0	4	6	4	0	0	0
40–44	7	5	2	0	0	0	6	8	1	1	1	0
45–49	3	6	5	2	0	0	5	5	2	1	0	0
50–54	5	6	0	2	0	0	1	10	5	0	0	0
55–59	1	7	0	1	0	0	6	4	2	3	1	0
60–64	0	6	6	1	0	1	3	4	4	0	2	0
65–69	0	4	4	1	2	0	3	1	3	0	0	0
70–74	0	2	3	1	3	0	3	6	1	3	1	0
75–79	1	0	2	1	0	0	1	2	4	2	0	0
80–84	1	0	0	0	0	0	3	1	2	0	0	0
85–89	0	0	0	0	0	0	0	0	0	0	0	0
90–94	1	1	0	0	0	0	1	0	0	0	0	0
Total	46	51	22	9	5	1	66	61	31	11	5	0

spouses to whom they were wed for but a short time. The effect of frequent marriage on population growth is difficult to assess, but since it is a constant we can ignore it.

The marital state of individuals on the atoll, as revealed in table 6, cannot be adduced as a cause for the decline. Ulithians marry early, although not exceptionally so. Girls marry at least 5 years sooner than boys, starting, for the most part, when they have reached the 15–19 age group. There are few individuals not living in a marital state. Beyond the age of 24 there are only 5 single males and two single females. Divorce is very common, but the number of persons living in a state of divorce is small, being 15 for men and 25 for women. The number of widowed individuals is not high, considering the proportion of old people, but it is interesting that the number of widows is more than 4

TABLE 6

Marital state of individuals by age groups (1949)

AGE GROUP	SINGLE		MARRIED		DIVORCED		WIDOWED		TOTAL		
	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂ + ♀
10–14	10	7	0	2	0	0	0	0	10	9	19
15–19	12	1	0	10	1	3	0	0	13	14	27
20–24	6	0	9	10	0	2	0	0	15	12	27
25–29	1	1	13	7	0	2	1	2	15	12	27
30–34	2	0	5	9	2	2	0	0	9	11	20
35–39	0	0	10	14	0	0	0	0	10	14	24
40–44	1	0	11	15	3	0	0	2	15	17	32
45–49	0	0	12	9	3	1	1	2	16	12	28
50–54	0	0	10	10	3	2	0	4	13	16	29
55–59	0	0	7	9	1	1	1	7	9	17	26
60–64	0	0	13	3	0	4	1	6	14	13	27
65–69	1	0	8	3	1	1	2	3	12	7	19
70–74	0	1	6	5	1	3	2	6	9	15	24
75–79	0	0	4	4	0	2	0	3	4	9	13
80–84	0	0	0	1	0	1	1	3	1	5	6
85–89	0	0	0	0	0	1	0	0	0	1	1
90–94	0	0	2	0	0	0	0	1	2	1	3
Total	33	10	110	111[1]	15	25	9	39	167	185	352

[1] The discrepancy between the total number of married men and women is due to the fact that one of the husbands has gone to live on Yap, without, however, having divorced his wife, who remains on Ulithi and is considered married.

times that of widowers. However, we must take into consideration that women of the age of 60 or over outnumber men 51 to 42—not a great difference, to be sure. Failure to marry, then, and failure to live in a married state, may not be considered causes of decline in population or the inability of the natives to effect a resurgence. There are only two women over the age of 17 who have never married. One is 25 years old and paralyzed in one leg; the other is 73 and psychotic. Of the men above the age of 17, thirteen have not married, but the majority of them are still in their twenties. It is interesting that informants insist people used to marry many years later than they do now. Should this be true, there is all the more reason to eliminate marital state as a factor in the depopulation.

Male absenteeism may have something to do with the low birth rate on Ulithi, but if so the effect has been very small. Able-bodied men used to go to work in German and Japanese phosphate mines on Angaur and Fais, so that for long periods of time they were away from their women. This is of course a new factor not previously operative under native conditions. It must to a large extent be minimized because of the prevailing attitudes towards extra-marital sex relations and illegitimacy. Along these lines it is interesting that the Harvard team studying depopulation on nearby Yap (Hunt *et al.*, '54) found that men with longer absences had a slightly larger number of recognized offspring than those with shorter absences. Even though the difference was not statistically significant it verifies my feeling that abstenteeism is a doubtful cause of lack of prolificacy on Ulithi. Another kind of absenteeism, resulting from the forcible impressment of most of the able-bodied adult male population into the labor service of the Japanese army on Yap around 1943-44, undoubtedly had a real influence in keeping down the birth rate because of the large numbers of men involved; but the effect was temporary and does not account for the previous decline.

As for the "loss of interest in life" theory popularized by Rivers ('22), it has no applicability to Ulithi, where the social system is still operating largely in autochthonous terms and the people display a great zest for life. Anyone who has visited the atoll can attest that there is no demoralization whatsoever. There is some drinking of fermented palm toddy, but my figures show that there are only one or two really heavy imbibers. Smoking is widespread among adults. No one has shown this to be a sign of demoralization. The practice, moreover, is an old one, for Carolinians have raised tobacco of their own for many years. Except for carpenter's tools and the like, there is no great envy of Western ways. Clothing remains almost completely native, and, it should be observed, is of healthful design. Houses are almost always of the old style and are kept clean and almost devoid of flies. While Christianity has made deep inroads since 1937, the effects, if anything, have been salubrious on morale. Ulithians who have been converted maintain it has created euphoria, and those who are still pagans show signs of wishing to embrace it. If in recent years, then, the size of families has decreased, it is not the result of an unconscious "race suicide" in protest against Europeans. So anxious are Ulithians to have children that they adopt them with eagerness, 45% of the people having been adopted either before birth or soon after. A fatherless child always has many potential adopters.

DISEASE AND DEPOPULATION

The one factor which has not been a constant among Ulithians is introduced diseases. It is my considered opinion that after Ulithi had been opened up to the outside world when the Germans assumed control in 1899, illnesses were introduced which subsequently had a highly detrimental effect on population numbers. Unfortunately, specific medical figures for Ulithi are virtually absent, for it has been the practice of the German, Japanese, and American governments to include Ulithi in general studies of the whole Yap area. Therefore, a good deal of what I shall have to say must be derived from indirect sources.

I wish to submit that gonorrhea alone has created enough havoc to merit a major role in the dispeoplement of the Carolines. The nefariousness of this disease is not that it kills but that it prevents women from conceiving. The impeding of impregnation is in the long run more injurious than death from the respiratory and other diseases apparently introduced in modern times. There is no way of knowing exactly when gonorrhea was introduced into the Carolines, but it does not seem unreasonable to assume that it became widespread after these islands had been opened up to the outside world by the Spaniards, and, later, the Germans and Japanese.

Unfortunately, no statistics are available to indicate the exact prevalence of gonorrhea on Ulithi, but in 1948 a medical officer of the U. S. Navy, during his tour of the atoll, stated to the author that his experience throughout the West Carolines showed that almost 80% of the adult population was afflicted with this disease. In his estimation, the percentage he named was possibly applicable to Ulithi, though he admitted that he had no figures and that his guess might be too high. This physician was of the firm conviction that the low birth rate on Ulithi and other islands of the area could be attributed to gonorrhea.

Pharmacist's mates attached to the LORAN station on one of the islands occasionally administer simple medical aid to natives and take vaginal smears. They, too, maintain that the rate of this venereal disease is unusually high. The natives themselves admit the strong prevalence of the disease, which they call *hasich*. It is regrettable that a thorough medical examination has never been made so as to provide us with precise figures, but there is no doubt in the mind of the writer that the proportion of persons infected by the gonococcus bacterium is inordinately high.

It is significant, in support of the gonorrhea theory, that when women and their husbands are suffering from the disease and they are treated with modern drugs, the birth rate increases. Reinfections, of course, readily occur, due to the prevalence of adultery and the lax regulation of premarital relations. But it is a matter of common knowledge among the natives that soon after a woman has submitted to western medicants for treatment she often bears a child, despite the fact that she may not have conceived for many years, if at all. In 1948, several men who had been sent to Palau for medical training returned to Ulithi and began a therapeutic program. Immediately, the number of pregnancies showed a spectacular increase, especially on the island of Falalop where treatment had been most intense. After I had left Ulithi in 1949 my former native assistant sent me some records of the births for that year and the following, and they pointed to a marked increase over previous years.

The gonorrhea theory of depopulation has had contrasting support. Figures published by the various governments in control of the Caroline Islands usually show it to be of moderate or even low incidence, but there is every reason to believe that examinations have been superficial and latent cases unobserved. At least one authority, however, whose publication I have not had an opportunity to examine, maintains that after an intense examination of 1,102 women on Yap, he found that 42.8% had gonorrheal vaginitis and endometritis, and that probably inflammations of the tubes and ovaries were also present. This same authority, a Japanese physician named Fujii, believes that the low birth rate on Yap has been due to this disease (Fujii, '34, cited by Hunt *et al.*, '54). Yap is a close neighbor of Ulithi and what applies there may also apply to its smaller dependency.

Respiratory diseases have taken a heavy toll in this century among the people of the islands. They have a great dread of influenza and bronchitis, which they feel are created by sorcerers sent over from Yap to punish them when they have broken taboos or failed to send tribute to their overlords (Lessa, '50). The effects of these diseases have aparently not been published specifically for Ulithi, but the records of the German and Japanese governments refer repeatedly to the high mortality resulting from them in the Yap administrative district, as well as in other parts of what is now known as the Trust Territory. Respiratory diseases seem no longer to be as fatal as they once were, but there is no doubt that they have had serious effects in the past. While emphasizing these infections, I believe that they are definitely subordinate to gonorrhea, for the major cause of depopulation on Ulithi is due simply to the failure of the women to become pregnant.

The large number of persons with paralyzed limbs bears testimony to the fact that there have been some severe epidemics of what seems to have been poliomyelitis, which struck soon after the Japanese administration began. Seven males and 5 females show paralysis, their ages ranging from 9 to 73, with a fairly even distribution in between. This means that 2.8% of the population shows signs of the disease, and when one considers that there may have been cases which did not survive the infection, and others which may have been of a non-paralytic type, it is obvious that we have here a force to contend with in the depopulation of the atoll.

Other diseases may be largely discounted. Dengue, filariasis, malaria, rickettsioses, typhus, typhoid fever, leprosy, and many other diseases are either absent or unimportant in this area. Syphilis is virtually nonexistent because, supposedly, of the prevalence of yaws, although the latter has been almost eradicated since the arrival of the American forces in 1944 (Wees and Thornton, '50). Tuberculosis, a serious problem in some parts of Micronesia, is not lethal on Ulithi. On one of the rare occasions in which the atoll is specifically mentioned in a medical report, it was discovered that in 1949 two persons examined by X-ray had "suspicious" cases of pulmonary tuberculosis, and one had tuberculosis of the lymph nodes. The people of Ulithi, according to this survey, which was made by the hospital ship, U. S. S. "Whidbey," in common with the other islands of the Yap district, seem to have considerable resistance toward tuberculosis. Tuberculin skin tests showed a high percentage of the people gave a positive reaction. Apparently tuberculosis has been overrated as a factor in mortality rates for this area (McNair *et al.*, '49).

Intestinal parasites, especially trichuria and hookworm, are common, although less so than on most islands of the Yap district (McNair *et al.*, '49). Hunt and his associates ('54) are of the opinion that although intestinal parasites may have slightly depressed the birth rate on Yap, it has not prevented some of the most seriously infected peoples of the world from having among the highest rates of population increase. In any case, to my knowledge these parasites are not a new factor on Ulithi and therefore must be discounted from that point of view, too.

TABLE 7

Age incidence of dental caries and tooth loss

AGE GROUP	NUMBER OF INDIVIDUALS				
	In Age Group	With Caries		With Lost Teeth	With No Caries or Lost Teeth
		Apical	Gingival		
16–19	7	5	1	0	1
20–23	7	2	0	1	4
24–27	6	1	0	0	5
28–31	5	1	0	1	3
32–35	2	1	0	2	0
36–39	6	1	0	3	2
40–43	4	2	0	1	2
44–47	4	0	0	3	1
48–51	5	1	0	4	0
52–55	3	2	0	2	0
56–59	4	0	1	3	1
60–63	3	0	1	3	0
64–67	2	1	0	2	0
68–71	1	0	0	1	0
Total	59	17	3	26	19

Finally, malnutrition must be eliminated as an operative factor in the depopulation of Ulithi. Macroscopic inspection shows the people to have the appearance of good health and physique, especially in the case of males, who are unusually muscular and strong, with few traces of fat even in old age. In a group of 59 adult males studied anthropometrically, only one man in the sample showed outright fatness (Lessa and Lay, '53). Table 7 indicates that if teeth are a measure of good health and nutrition, Ulithians surely rate high. Of the group of 59 men studied, almost one third had all their teeth and no caries. The highest incidence of caries is in the youngest age bracket, possibly reflecting deleterious contacts with military and other American installations during and after the war. According to the opinion of a civilian Navy dentist working on the atoll in 1948, tooth loss on coral atolls appears to be due more to pyorrhea resulting from heavy tartar deposits than to caries. This view seems plausible enough, for there is considerable calcium in the food and it creates deposits which are greatly irritating to the gums.

THE FUTURE

It now seems likely that the population of Ulithi will show a sharp upturn. Young girls have recently been trained in simple medicine and obstetrics, and the number of young men with medical training has reached a fair size. The U. S. government gives regular medical and dental treatment. Men no longer are removed from home in order to work abroad. Travel on the seas is almost entirely in modern vessels. The deleterious effects of typhoons have been removed by the ready assistance of American authorities, so that no one need starve. The author's prediction is that, if a strong health program is maintained and if special circumstances do not intervene, there may eventually be so many people on the atoll that some sort of control will be necessary. The possibility of overcrowding will not, however, be realized in the near future. Ulithi can support twice its present population, but it will take many years to reach that condition. The bare realities of the matter are this: The percentage of Ulithians below the age of 15 is next to the lowest of any of the peoples of Guam, Samoa, and the Trust Territory of the Pacific Islands for which data are at hand. This may readily be seen by consulting table 8, which shows the percentage of individuals of the age of 14 or less in the above-mentioned places, together with, for comparison, the United States, England and Wales, and Japan. So low a proportion of children if sustained is unhealthy from the point of view of survival.

Moreover, the number of persons 50 years of age or older is exceptionally high, 32.4% of the total. This may be compared with the following percentages for fast-growing populations: Saipan Chamorros, 8.9; American Samoa, 9.2; Guam, 10.3; Japan, 15.5. Of course, a high proportion of post-reproductive individuals does not in itself indicate an unhealthy situation. In 1949 in the United States, for example, 19.6% of the people were in the post-reproductive category. It is when the old age ratio is high and the birth rate is low that the situation, as in Ulithi, becomes ominous.

There is enough room to expand, not only back to the high level of 1904 but even more. The density of population per square mile was 442.8 in that year and was only 233.9 in 1949. These figures are low when compared with some of the more densely populated atolls of the Carolinian area: Eauripik, 1633.3; Fauraulep, 750.0; Lukunor, 768.8; Losap, 1866.7; Namoluk, 734.4; Satawan, 782.4; Murilo, 716.0; Pingelap, 916.2, and Kapingamarangi, 930.8 (U. S. Navy, '51). There are several islands in the Ulithi group that were once occupied but later abandoned, and there are others that apparently have never been inhabited but could hold many families. Some of the abandoned islands were vacated upon orders of the Japanese, who wished to reserve them for raising copra, but Ulithians never have felt the need to reoccupy them even when at liberty to do so.

TABLE 8

Percentage of individuals of pre-reproductive age (under 15) in various places[1]

PLACE	PERCENTAGE
Outer Islands, Palau District (1948)	9.9
Ulithi (1949)	20.9
England and Wales (1948)	21.4
Yap (1947)	23.7
United States (1949)	27.1
Marshall Islands (1948)	30.0
Truk (1947)	32.2
Ponape (1948)	33.0
Japan (1948)	34.7
Palau (1948)	36.0
Saipan Carolinians (1946)	36.6
Guam (1940)	44.1
Saipan Chamorros (1946)	45.1

[1] Sources: Lessa (present study); United Nations ('50); U. S. Navy ('48).

SUMMARY

In 1904 it was reported by the German government that the population of Ulithi stood at 797. A detailed census taken in 1949 by the author showed a marked drop to 421, with a large proportion of old people and a small proportion of young ones.

Various possible causes for this decline are examined and rejected as being inapplicable, especially because most of them are not new factors and therefore cannot explain a new trend. Among them are: typhoons, shipwrecks, abortion, infanticide, contraceptives, infrequency of coitus, inbreeding, marital state, male absenteeism, and loss of interest in life. In connection with the last-named it should be observed that in 1949 Ulithian society was still operating largely in unacculturated terms and that native institutions were for the most part very vigorous.

Diseases introduced by Europeans seem to be the real reason for the decline. Of these, gonorrhea is the most important. This accords with the fact that the women of Ulithi do not become pregnant with sufficient frequency to enable the population to increase, or, at least, to maintain itself. Respiratory diseases too are very injurious to population growth. Osteomyelitis may be another major contributory cause. Other diseases, particularly tuberculosis, syphilis, trichuria, hookworm, and malnutrition, do not apply.

Beginning about 1948 the use of medicants to treat gonorrhea has apparently been responsible for a noticeable increase in pregnancies, and it is possible that the atoll will now witness a marked upturn in the population curve. If this should occur, there is ample room for almost a doubling of the population.

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TECHNOLOGICAL ADVANCE AND MAJOR RACIAL STOCKS[1]

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WE HUMANS are a polytypic species. Both as individuals and as breeding populations, we differ from one another in numerous biological characteristics. The extent to which these differences are due to environmental influences during the growth of each individual has not yet been determined. It remains clear, however, that genetic differences do exist between the various tribes of men, and, naturally enough, attempts have been made to find some order in the variety. Most typically, scholars have attempted a three-fold division of our species: after all, three has long been a sacred number in our culture. So we continue to hear of Mongoloids, Negroids and Caucasoids as basic, primary or original stocks, from whom all of us today, by further evolution or hybridization are derived.

Some authorities, like Howells ('44), insist upon 4 rather than three; some, like Boyd ('50, pp. 268-272), speak of 6. Sergi ('11) divided us all into two original varieties, the Eurafrican and the Eurasiatic. Keith ('49, pp. 234-244) has written also of two basic groups, one being deeply pigmented or Negroid, the other being rather less pigmented but variable. Three is, however, the more orthodox number, and it must be noted that each is named after a group or type which is very numerous at our stage of history. Average or run-of-the-mill specimens of each stock, as described by the usual authorities, can also be recognized by anyone with our cultural perceptions as being different from either of the others.

Hooton's position with respect to the existence and primacy of the three major stocks is made clear by his statement in *Up From the Ape* ('46, p. 449): "The differences between the various subgroupings within the three great or primary races are *obviously* more recent, and quantitatively and qualitatively *less* than exist between the primary races respectively." Kroeber ('48, p. 134) accepts this idea, albeit with some reservations. He refers, for instance, to "remaining minor, aberrant forms which are best kept separate, provisionally. Some, like the Ainu and Australoid, appear to affiliate preponderantly with one of the three great classes." Ashley Montagu ('51, pp. 301, 304-306), on the other hand, apologizes for mentioning divisions or major stocks. Yet he goes on, at once, to speak of Nilotes as "probably hybrids between Hamites," whom he classifies as a branch of Whites and "Forest Negroes." He supposes a group whom he labels "Half Hamites" to have arisen in the same manner. Why, therefore, he distinguishes them from Nilotes he does not say.

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It is, indeed, the existence of such groups as Nilotes and Ainu, the extraordinary distribution of dark-skinned pygmy types, the accumulation of findings concerning genetic characteristics, and the recent studies of climatic adaptations within the human stock, which lead me, at least, to doubt the existence, past or present, of two, three or 4 major stocks, or even of the 6 listed by Boyd ('50) from his study of blood-type distributions. Coon, Garn and Birdsell ('50, p. 115), indeed, state, that "a stock is not a race, but merely a convenience in classification: It is a lumping of races which seem similar to each other in broad categories." In fact, I wonder if the concept of the major stock does not usually serve to mislead the student of taxonomy, which is an inconvenience rather than a help. From the concept of the major stock flows, perhaps sometimes unconsciously, the assumption of genetic priority, which is explicitly accepted by Hooton.

Is this assumption well based? What is the evidence? Let us take a few cases.

The data of human genetics provides evidence of a remarkable difference in numerous characteristics within each of the major stocks. This is notably demonstrable in comparative serology. Some of the differences in gene-frequency do not correspond with the lines which are supposed to divide stocks. Boyd ('50, pp. 268-269), consequently, would separate the Basques from the Caucasoids, and the American Indians from the Mongoloids. The more we learn of human genetics, the greater the number of genetically distinctive groups we discover.

The data of palaeontology provide far better evidence concerning the regional antiquity, at least, of Bushmen (Galloway, '37), Australoids (Weidenreich, '45), Nilotes (Leaky, '35), Ainu (Hooton, '46, p. 402) than of Mongoloids and Negroids. Regional variety within the Caucasoid group appears to be as old as the group itself. The best fossil relics point to what Hooton calls sub-races or composite races rather than to the great primary races which are said to be the ancestral stocks.

The data from ethnology concerning the geographical distribution of living and recently living peoples provides most equivocal evidence. Although the Caucasoid stock appears to have its major center of distribution in Europe and the Middle East, it is alleged to have contributed genes to such groups as Ainu, Polynesians and Hottentots. Negroids are scattered, mostly in tropical areas, from Fiji and Tasmania to Senegal and Capetown, Negritos sporadically distributed among them. There are large gaps, by oceans, deserts, and intervening populations of quite different phenotype and genotype, which separate the dark-skinned tribes. Those who accept, even as a convenience, the prior existence of Negroids or Caucasoids as major stocks have had to exercise much ingenuity to explain these improbable distributions.

Kroeber ('48, p. 125), for instance, in writing of the Ainu, assumes that if they resemble Europeans (as he appears to think they do) they must be a *branch* of the Caucasian stock, they must have come from the West, and that they have no connection with the Mongolian Japanese among whom they now live. Further unstated assumptions which must logically underlie these statements are: that the Europeans are typical Caucasians, that the Japanese are typical Mongolians, that there is some other direction from which to reach Hokkaido and that the Ainu have always been, as they are now, a relatively small population. None of these unstated basic presuppositions can be demonstrated to be true, and, indeed, most of them are rather unlikely. The Ainu resemble native Australians at least as much as they resemble either European or Asiatic Caucasians. Japanese are not especially typical Mongolians. There is no particular reason to believe that the relative proportions now existing between the various racial and tribal groups, in numbers, have existed unaltered throughout prehistory.

Let us look again at the Nilotic tribes. If indeed they are nothing but hybrids between Negroids and Caucasoids one would expect them to be intermediate in genotype between the original stocks. As a matter of fact they possess a notably high incidence of the allele for sickle cells, a low incidence in the allele for blood-group A, and a typically African incidence in the alleles for the Rh subtypes (Mourant, '54). One might expect them to be quite variable in phenotype, at least in skin color, if they are hybrids, but one finds them to be especially dark-skinned, and elongated in body-build far more than either of the presumptive ancestral stocks, Negroid or Caucasoid. In other words, they have numerous characteristics which can not be explained by the hypothesis of mixed ancestry.

Are they, on the other hand, more closely related to all other very dark skinned tribes, no matter where, than they are to less deeply pigmented peoples to the north, such as Caucasoids of the Mediterranean area? There is positively no indication or even suggestion from genetics that this might be so. We have no record of sicklemia in Melanesia, for instance, but the gene is known among Greeks, Sicilians and some tribes in India. Although blood-group A is very common among Melanesians, the subtype A_2 is unknown: both among Nilotic and Mediterranean peoples the ratio of A_2 to A_1 is high. The allele frequency of N is high in Melanesia, but much lower throughout Europe and Africa. The Melanesians are utterly un-African in Rh subtype distribution (Mourant, '54). Indeed, it seems to be reasonably clear that a considerable variety of very dark-skinned peoples exist, and that they are not necessarily closer cousins to one another than any of them are to less dark-skinned groups.

In fact, there are numerous peoples in the world whose characteristics simply can not be adequately explained by any of the theories derived from a belief in two, three or 4 original major stocks. Bushmen, Basques, Lapps, even Polynesians and American Indians just don't fit. Nevertheless, there they are, alive and breeding just like the rest of us. If one assumes that the major stocks are original, and the actual people of the world just a later derivation, great difficulties are created.

Now as a matter of fact people don't live, work and breed in racial groups, but in tribes, castes and other culture-bound groupings. Opportunities for spreading one's genes in distant places have, in the past, been limited. Most migrants move in groups, rather than individually. The social group, furthermore, modifies the natural or raw environment to a greater or lesser degree, depending upon its technology and social organization. Some forms of technology and social organization permit and encourage greater population densities than do others.

The invention of Neolithic ways of life was revolutionary in this respect (Childe, '36). With agriculture, the population of a tribe could increase rapidly, and we have ample archaeological evidence to show that population movements followed this increase, as farmers sought new lands to cultivate. Areas previously rather useless to man could now be profitably exploited. Other technological advances have permitted the survival of individuals without climatic adaptation. Indeed, the better the technology, the higher the survival rate, no matter what the bodily form. Members of European royal families can even afford to have hemophilia.

Today the vast bulk of the world's population, although not all, can be squeezed somehow into one or another of the so-called major stocks. In seeking to uncover the story of ancestral relationships within the human stock, however, we should consider, instead, the most likely distribution of population before the Neolithic revolution. After all, that is less than 10,000 years ago, only 1% of the period of man's existence. The population explosions which resulted in the present distribution occurred only in limited areas at first, and, consequently, affected only a limited number of the population groups at the time. Yet these groups gained an initial advantage in numbers, and such an initial advantage is difficult to overcome. The genetic systems of populations which happen to have good tools are not better, in any sense of the word, than are the genetic systems of populations which lack good tools. The former do have a higher survival value than the latter, however, simply because the tools help their owners to survive and to multiply.

Now we can not, of course, reconstruct with any high degree of precision, the picture of the distribution of the world's population at the end of the Palaeolithic. Nevertheless, the advance of archaeological investigation and of ecological studies do permit us to come to logical conclusions with respect to relative sizes of populations inhabiting certain areas of the world in the days when all were food-collectors.

Before the beginning of the Neolithic, real forests, whether tropical jungle or temperate zone, were not especially desirable places for human habitation. From what we know of hunting and fishing peoples, we have every reason to believe that parklands, savannahs, prairies and the like can support denser populations, because of greater abundance of seeds and game and greater ease of collection and hunting. Consequently, we may be sure that Europe was not thickly inhabited, and the archaeological evidence suggests that, with new technologies, population movements from the Near East repeatedly came into Europe.

On the other hand, East and South Africa, then as now, appear to have been, like the Near East, land fit for hunting and collecting. It also appears to have been until even later, indeed much later, inhabited at least in part by Khoisans, or Boskopoids, presumptive ancestors of Bushmen. There is little or no reason to suppose that East and South Africa, home of the ancestors of Bushmen, were less densely populated than the Near East, home of the ancestors of modern Europeans. In area, the parklands, savannahs and prairies of the Near East, of what is now the Moslem world, from Pakistan to Morocco, do not exceed 2,500,000 sq. miles. Regions similarly favorable to hunting-collecting peoples, from an ecological standpoint, in East and South Africa cover an approximately equal area. Consequently, there is no reason to suppose that populations with genetic systems similar to those of Europeans were, 10,000 years ago, more numerous than were populations with genetic systems similar to those of Bushmen. Yet today the first is regarded as the typical representative of a major, primary stock, the latter as an aberrant and mysterious racelet.

Are we not fooling ourselves, in a case like this, simply because of the present day preponderance of numbers?—a preponderance due simply to the fortuitous correlation between Caucasoid types and the invention of agriculture and the technological developments which later, and doubtless consequently, ensued in history.

This, of course, is even more true if we consider the blondish varieties of Europeans. Many suspect that really extensive and widespread depigmentation is an adaptive character suitable to the climate of northwest Europe (Coon, Garn and Birdsell, '50, p. 50). It may very well be exceedingly ancient there. But this region, until only 5,000 years ago, was most sparsely inhabited by primitive savages. They are quite likely to have numbered only a few tens of thousands, if we can judge by population densities of peoples of equivalent economies and environments among today's hunting and fishing groups, such as the Yahgan. Yet we unconsciously designate such Europeans as the standard Caucasoids, simply because there are now some hundreds of millions of people with such physical characteristics. Let us examine how this came about: here is one case concerning which we have ample historical documentation.

By the end of the Middle Ages, the population of the British Isles, the Low Countries and Scandinavia was still less than 10,000,00C. Because of geographic position and historical circumstance, the British emigrated in astonishing numbers, settling in lands previously only scantily occupied. Although in 1600 there were perhaps 3,000,000 there are now at least 150,000,000 of British stock. The world population has increased by 6 times in these last few centuries, that of British ancestry by 50 times. This is no sign of superior British virtue, mentality or fecundity, yet it is an evolutionary fact. Areas in Europe containing a high per cent of blondism held perhaps 3% of the world's inhabitants then. People originating from such areas comprise at least 12% of the world's inhabitants now. If the allele or alleles for blondness have increased proportionately (and, with modern technologies, blonds can survive even in the tropics, especially if they are in the upper class) blondness should now be more than 4 times as common as it was then. Technology, and historical events resulting from technology, have produced a remarkable change in the incidence of this allele. This is evolution in action.

The population explosion in part of the areas inhabited by Caucasians, remarkable though it has been, is not unique. Neolithic techniques, although apparently developed and originated very largely in areas occupied by people of this population group, have not been their unique possession by any means.

In China, for instance, food production took root long ago, with the result that the local population there began to expand also, with respect both to numbers and area occupied. The genetic characteristics of the Chinese, like those of the Europeans, have become much more common than they were in the Palaeolitic. The movement southward of peoples in East Asia is amply documented by ethnological distribution studies, linguistics, archaeology, history and indeed by contemporary events. Extensive hybridization, and migration as well, have followed culture-trait diffusion, so that both phenotypes and genetic systems typical of northeast Asia have spread more and more to the south, even into Indonesia.

As a result of this process, which has almost certainly taken place only since the application of Neolithic, or even Iron Age techniques, we find hundreds of millions of individuals with more or less Mongoloid characteristics spread along the western rim of the Pacific basin, and, scattered among them, in all sorts of refuge areas, populations which are clearly much less Mongoloid. At the end of the Paleolithic, the situation must have been far different, with few Mongoloids living in the open lands to the north, and a great variety of populations with genetic systems quite unlike that of the Mongoloids in Japan, China, Indo-China and Indonesia. This proposition is so widely accepted that it need not be argued here, yet its implications with regard to the question of the genetic priority of major stocks seem to have been neglected.

It seems to me that we can be quite certain that 10,000 years ago the numerical proportions between populations of different genetic characteristics must have been violently different from what they are today. We do not yet have adequate data to postulate the typical characteristics of most of the tribal groups then in existence. But the numbers of tribes with genetic systems similar to the so-called Mongoloid and Caucasoid stocks must have been relatively few. Blonds were probably as rare then as Bushmen are today. The numbers of Bushmen, of Australoids, and perhaps of other populations as well, was relatively great, but probably exceedingly diverse genetically.

Changes in proportion, which have led us to attempt classification into three major stocks, have come about during the last 1% of human existence and have come about entirely because of a fortuitous correlation between more advanced technical skills and certain racial characteristics. Historical circumstances, not racial virtues, determined the expansion of a certain few among the many population groups which existed at the beginning of the Neolithic. When man succeeds in modifying his environment he directs the future course of his own evolution, albeit quite unaware of his direction, his speed, or even of the fact that he is in the driver's seat.

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THE NATCHEZ INNOMINATE BONE

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On 6 October 1846, at a meeting of The Academy of Natural Sciences of Philadelphia,

> Dr. Dickeson exhibited a large and remarkably varied series of fossil bones, obtained by him from the vicinity of Natchez, Miss. The collection embraces the entire head and half of the lower jaw of *Megalonyx Jeffersoni*, now for the first time discovered; together with many parts of the skeleton, and indeed of several skeletons of that animal, sufficient to enable its complete osteological reconstruction. The stratum that contains these organic remains is a tenacious blue clay that underlies the diluvial drift east of Natchez, and which diluvial deposit abounds in bones and teeth of the Mastodon giganteum.
>
> The animals associated with the *Megalonyx* are, an *Ursus*, a *Bos*, two species of *Cervus*, one or two species of *Equus*, and several undetermined genera, all which are now in progress of delineation and description for the Academy's Journal. [The fossils actually discovered were *Mastodon americanus*, *Megalonyx jeffersoni*, *M. dissimilis*, *Ereptodon priscus*, *Mylodon harlani*, *Equus major*, and *Bison latifrons*].
>
> Dr. Dickeson presented another relic of yet greater interest; viz., the fossil *os innominatum* of the human subject, taken from the above mentioned stratum of blue clay, and about two feet below the skeletons of the *Megalonyx* and other extinct genera of quadrupeds.
>
> This ancient relic of our species, is that of a young man of about sixteen years of age, as determined by its size and form, and by the fact that the epiphyses have separated from the tuberosity of the ischium, and from the crista of the ilium.[1] Nearly all the os pubis is wanting, the upper posterior part of the ilium is broken away, and but half the acetabulum remains. That this bone is strictly in the fossil state, is manifest from its physical characters, in which it accords in every respect of color, density, &c., &c., with those of the *Megalonyx* and other associated bones. That it could not have drifted into the position in which it was found, is manifest from several facts: 1. That the plateau of blue clay is not appreciably acted on by those causes that produce ravines in the superincumbent diluvial; 2. That the human bone was found at least two feet

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[1] This is incorrect. The bone is that of an adult.

> below those associated skeletons of the *Megalonyx*, all which, judging from the opposition, or proximity of their several parts, had been quietly deposited in this locality, independently of any active current or other displacing power; and lastly, because there was an admixture of diluvial drift with the blue clay, which latter retains its homogeneous character equally in the higher part that furnished the extinct quadrupeds, and in the lower part that contained the remains of men. Dr. Dickeson has announced his intention of returning, at an early period of the present autumn, to resume his explorations in this prolific and most interesting locality; and it is earnestly hoped that his researches may lead to a further elucidation of this important question in science.

In this manner was announced the discovery of what has since come to be known as "the Natchez pelvis." At the meeting of the Academy held 2 November 1847 "Dr. Dickeson made some observations on the mode of compression of the cranium in use among the Choctaw Indians, and the supposed object of the same." Thereafter Dr. Dickeson vanishes from the stage and is heard from no more.

Fortunately, but a few months before he gave the account of the Natchez innominate at the Academy of Natural Sciences, Dr. Dickeson had shown both the site and the bones to Charles Lyell, the English geologist, then visiting the United States. From his letters (1881) it would seem that Lyell was in Mississippi sometime during the first three months of 1846. In the narrative of his visit to the United States Lyell (1849), recalling these recent events, writes:

> In company with Dr. Dickeson and Colonel Wailes, I visited a narrow valley, hollowed out through the shelly loam recently named "the Mammoth ravine," from the fossils found there. Colonel Wiley, a proprietor of that part of the State of Mississippi, who knew the country well before the year 1812, assured me that this ravine, although now seven miles long, and in some parts sixty feet deep, with its numerous ramifications, has been entirely formed since the earthquake. He himself had ploughed some of the land exactly over one spot which the gully now traverses.
>
> A considerable sensation was recently caused in the public mind, both in America and Europe, by the announcement of the discovery of a fossil human bone, so associated with the remains of extinct quadrupeds, in "the Mammoth ravine," as to prove that man must have co-existed with the megalonyx and its contemporaries. Dr. Dickeson showed me the bone in question, admitted by all anatomists to be part of a human pelvis, and being a fragment of the *os innominatum*. He felt persuaded that it had been taken out of the clay underlying the loam, in the ravine above alluded to, about six miles from Natchez. I examined the perpendicular cliffs, which bound a part of this water-course, where the loam, unsolidified as it is, retains its verticality, and found land-shells in great numbers at the depth of about thirty feet from the top. I was informed that the fossil remains of the mammoth (a name commonly applied in the United States to the

> mastodon) had been obtained, together with the bones of some other extinct mammalia, from below these shells in the undermined cliff. I could not ascertain, however, that the human pelvis, had actually been dug out in the presence of a geologist, or any practised observer, and its position unequivocally ascertained. Like most of the other fossils, it was, I believe, picked up in the bed of the stream, which would simply imply that it had been washed out of the cliffs. But the evidence of the antiquity of the bone depends entirely on the part of the precipice from which it was derived. It was stained black, as if buried in a peaty or vegetable soil, and may have been dislodged from some old Indian grave near the top, in which case it may only have been five, ten, or twenty centuries old; whereas, if it was really found in situ at the base of the precipice, its age would more probably exceed 100,000 years, as I shall endeavour to show in a subsequent chapter. Such a position, in fact, if well authenticated, would prove that man had lived in North America before the last great revolution in the physical geography of the continent had been accomplished; in other words, that our race was more ancient than the modern valley, alluvial plain, and delta of the Mississippi,—nay, what is more, was antecedent to the bluffs of Port Hudson and Natchez, already described. Now that elevated freshwater formation, as I shall by and by endeavour to show, is the remnant of a river-plain and delta of extremely high antiquity; and it would follow, if the human race was equally ancient, that it co-existed with one group of terrestrial mammalia, and, having survived its extinction, had seen another group of quadrupeds succeed and replace it.

The good judgment and great authority of Lyell very properly served to put the Natchez innominate into the suspense account. But from the beginning there were objections to this. In a most able appendix on "Geology and palaeontology in connection with human origins," contributed to Nott and Gliddon's (1854) volume, Usher wrote:

> One human pelvis found near Natchez, by Dr. Dickeson, is an undoubted fossil; yet we are told that ferruginous oxides act upon an *os innominatum* differently than upon bones of extinct genera lying in the same stratum, lest natural incidents might give to man, in the valley of the Mississippi, an antiquity altogether incompatible with received ideas: and Sir Charles Lyell accordingly suggests a speedy solution of the difficulty by saying that a fossilized *pelvis* may have fallen from an old Indian grave near the summit of the cliff.

In his book on the antiquity of man Lyell (1863) returned to a discussion of the Natchez innominate. Here Lyell points out that there had been an earthquake in the region around Natchez in 1811-12 and the earth, in consequence, had been much fissured.

> One of the narrow valleys near Natchez, due to this fissuring, is now called the Mammoth Ravine. Though no less than seven miles long, and in some parts sixty feet deep, I was assured by a resident proprietor, Colonel Wiley, that it

> had no existence before 1812. With its numerous ramifications, it is said to have been entirely formed since the earthquake at New Madrid. Before that event Colonel Wiley had ploughed some of the land exactly over a spot now traversed by this water-course.
>
> I satisfied myself that the ravine had been considerably enlarged and lengthened a short time before my visit. and it was then freshly undermined and undergoing constant waste. From a clayey deposit immediately below the yellow loam, bones of the *Mastodon Ohioticus*, a species of megalonyx, bones of the genera *Equus*, *Bos*, and others, some of extinct and others presumed to be of living species, had been detached, and had fallen to the base of the cliffs. Mingled with the rest, the pelvic bone of a man, *os innominatum*, was obtained by Dr. Dickeson of Natchez, in whose collection I saw it. It appeared to be quite in the same state of preservation and was of the same black colour as the other fossils, and was believed to have come like them from a depth of about thirty feet from the surface. In my "Second Visit to America," in 1846, (Vol. ii, p. 197) I suggested as a possible explanation of this association of a human bone with remains of a mastodon and megalonyx, that the former may possibly have been derived from the vegetable soil at the top of the cliff, whereas the remains of extinct mammalia were dislodged from a lower position, and both may have fallen into the same heap or talus at the bottom of the ravine. The pelvic bone might, I conceived, have acquired its black colour by having lain for years or centuries in a dark superficial peaty soil, common in that region. I was informed that there were many human bones, in old Indian graves in the same district, stained of as black a dye. On suggesting this hypothesis to Colonel Wiley of Natchez, I found that the same idea had already occurred to his mind. No doubt, had the pelvic bone belonged to any recent mammifer other than man, such a theory would never have been resorted to; but so long as we have only one isolated case, and are without the testimony of a geologist who was present to behold the bone when still engaged in the matrix, and to extract it with his own hands, it is allowable to suspend our judgment as to the high antiquity of the fossil.

Vogt (1864) reproduced Lyell's 1849 account of the Natchez bone in his *Lectures on Man*.

Leidy (1869) states "The specimen may have been cotemporary (*sic*) with the remains of extinct animals, with which it is said to have been found, though it appears to me equally if not more probable that it may have fallen into the formation, from an Indian grave above, at a comparatively recent date, and became stained like the true fossils, from ferruginous infiltration."

Twenty years later Leidy (1889) repeated the same view.

Schmidt (1872), who had examined the Natchez innominate and measured it, presented a detailed analysis of the geological evidence, and concluded in favor of the high antiquity of the bone. He pointed out that the bone is really that of an adult, but so damaged that it resembles the innominate of an immature individual. Disagreeing with Lyell he considered the bone to date from the Champlain epoch.

> Fragen wir nach dem Alter des Natchez-Fossils, so egiebt sich, dass ein ganz beträchtliches sein muss; sein Zeit fällt in die Champlain-Epoch, welche, wie wir früher sahen, der grössten Ausdehnung der Gletschen in Amerika folgt. Das Mastodon, Megalonyx und ein ganz Reihe anderer postteriärer, jetzt ausgestorbenen Thiere noch mit dem Menschen und dem Besitz des Landes.

It has been long believed that the exact location of the Natchez site has been lost to human knowledge. Thus Sellards (1852) writes, "If the exact location from which Dickeson originally obtained the fossils can be re-located, valuable additional discoveries might be made." Actually the fairly exact description of the location of the site has been in print since 1873. In that year Foster (1873) gave an account of the Natchez find, and went on to introduce some "MS Notes, committed to the Author," writing as follows:

> In this conenction I would introduce the testimony of a highly competent observer, Porfessor C. G. Forshey. He states:
>
> "I examined the spot where the bone was found—in Berbard's Bayou, just above the bridge, on the Pine-ridge road, two and one-half miles from Natchez. The material from which it was taken was a dark loam in the bottom of the thirty-feet bayou. It was probably not *in situ*, but this loam, and the bone too, probably had caved in from some point above, and had been drifted thither from fields of several miles square, above the road. A dozen plantation burial places, and Indian mounds and camps had been exposed above for centuires; and in recent years, since inhabited by the whites (for a hundred years), the drains had cut through the surface to the depth of twenty and even forty feet in the bluff loam-beds. The probabilities are a hundred to one, that this bone was not of the Bluff (mastodon) formation, but of the recent era.
>
> "The mastodon bones and all others, of which there were many, whelmed in the floods which deposited the bluffs, were all rotten. We had the utmost difficulty in preserving any of them—tusks or bones, and only succeeded when we had varnish or glue at hand to protect them. No human bones of any kind were ever found, unless this pelvic bone is an exception."

Forshey had apparently made extensive studies of the geology of the Mississippi basin. Lyell refers to him several times in the 1850 edition of his *Principles of Geology*. Lyell refers to "Mr. Forshey, an eminent engineer . . ."

In the 'nineties Thomas Wilson of the National Museum at Washington became acquainted with the method of dating bones by fluorine analysis through reading Carnot's 1893 paper on that subject, and in that year submitted for analysis samples of bone taken from the Natchez innominate and from an associated *Mylodon harlani* to Professor R. L. Packard, who was then working in the laboratory of the Museum. The samples from the human bone proved inadequate for analysis, and it was then that the large hole was produced in the iliac blade by removing the button of bone from it. The second test proved satisfactory, and in 1895 Wilson published the results. The fluorine and anhydrous phosphoric acid determinations obtained by Packard were as follows:

	%F	%P_2O_5
Natchez human pelvis	0.38	20.77
Natchez *Mylodon harlani*	0.28	26.59

Wilson submitted samples of the same bones to Mr. Hildebrand, "a chemist of the United States Geological Survey, on duty at the National Museum, who furnished the following results of his analysis."

TWO FOSSIL BONES

		Man Per cent	*Mylodon* Per cent
Loss at 100°C		4.55	6.77
Loss on ignition		16.54	21.18
Silica	(SiO_2)	22.59	3.71
Phosphoric acid	(P_2O_5)	17.39	23.24
Alumina	(Al_2O_3)	3.21	4.02
Iron protoxide	(FeO)	5.65	4.44
Manganese protoxide	(MnO)	1.65	3.40
Lime	(CaO)	25.88	30.48
Magnesia	(MgO)	0.95	0.78

From these findings Wilson (1895, p. 305) concludes "The importance of this analysis will be apparent at a glance. The human bone is in a higher state of fossilization than is that of the mylodon." Wilson's overall conclusion from the evidence of these analyses is "(1) that according to the averages made by Mons. Carnot, the bones under present consideration, the man and the Mylodon, are substantially of the same antiquity, and (2) by the same comparison their antiquity is about midway between the modern bones and those of the Quaternary geologic epoch" (p. 725).

At about the time that Wilson became acquainted with Carnot's paper Brinton delivered himself of some admirable cautionary remarks which all modern workers would do well to consider. Brinton (1893) wrote:

> It seems to me . . . that this test, which I learn about from an abstract in the Journal de l'Alliance Scientifique, March 15, is open to some serious risks. Not only do the inorganic constituents of bone differ largely in the different osseous tissues of the same skeleton, but they notoriously vary at the different epochs of life. According to the analyses of Heintz, the fluoride of calcium in the average femur of an adult is about 3.5 of its inorganic constituents. Where the proportion in ancient bones differs notably from that in modern, how can we decide what part of it is owing to post mortem changes conditioned on the quality of the soil, the amount of percolation, the length of exposure before inhumation, and the like incidents? While it would be most desirable to have at hand a positive chemical test of antiquity, we must hesitate to accept as conclusive one which seems exposed to be influenced by these precarious conditions.

Hrdlička (1907) dismissed the Natchez bone as "a defective right os innomination, which, on comparison with a similar recent Indian bone, shows nothing peculiar. This is really all that can be said regarding it, and it would be quite useless to speculate as to its antiquity."

Most subsequent references to the Natchez innominate have all been to its equivocal status. The Wilson report had been completely overlooked, until it was rediscovered by Stewart in 1951. Before that time, however, and without any knowledge whatever of the work of Wilson, I decided to submit samples of both the Natchez human innominate and of the associated Mylodon to the fluorine test. With the kind cooperation of Dr. Horace Richards of the Academy of Natural Sciences of Philadelphia, in the Spring of 1948 I took with me to England samples of these two bones, and with the generous assistance of Dr. K. P. Oakley these samples were submitted to Mr. C. F. M. Fryd of the Department of the Government Chemist at London. The Natchez human sample proved insufficient for fluorine analysis, whereupon Dr. Richards most generously forwarded a larger sample of the human bone to London. This delay together with the accumulation of much pressing work in the London laboratory, prevented the analysis from being undertaken until recently. The results became available on 16 March 1954.

	%F		$\%P_2O_5$	
	Wilson	Present Study	Wilson	Present Study
Natchez innominate	0.38	0.88	20.77	26.2
Mylodon harlani	0.28	0.99	26.59	20.1

When we place these recent determinations by the side of those of Wilson, it will be seen that there is a pretty good agreement between the two. It being understood that variations between 20% and 26% P_2O_5, and between 0.2% and 0.4% F might well be found within a single specimen. For example, two portions of an antler from the Swanscombe gravels gave these results: %F 2.6, 2.2, %P_2O_5 30, 34.5.

The results of the chemical tests which have so far been applied to the human and extinct *Mylodon* bones indicate that they are of the same or similar age. Bearing all the necessary cautions in mind it may, then, be concluded that it is probable that the Natchez innominate and that of the extinct ground sloth *Mylodon harlani* found in association with it are of the same or similar age.

SUMMARY

1. An historical survey of the literature relating to the human innominate bone found at Natchez in 1845 presents the evidence relating to the antiquity of the bone.

2. A new study of the fluorine and anhydrous phosphoric acid content of the human ($F = 0.88\%$, $P_2O_5 = 26.2\%$) and associated *Mylodon harlani* ($F = 0.99\%$, $P_2O_5 = 20.1\%$) is reported.

3. The results of the chemical tests reported by Wilson in 1895 and those of the present study are in pretty fair agreement, and indicate that the Natchez human and *Mylodon* bones are of the same or similar age.

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LIFE EXPECTANCY IN THE CITY OF CHICAGO 1880–1950

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ALTHOUGH extensive contemporary mortality data are available, no historical series of comparable life tables has been constructed for large cities. Such a series of life tables for the City of Chicago is presented here. These life tables are of interest not only because they describe the dramatic decline in mortality rates during the past 70 years, but also because life table values may be used to construct generation life tables which give a picture of the mortality experience of an actual population rather than the customary synthetic population described in most life tables.

We shall begin by tracing the great increase in expectation of life which has taken place in Chicago during the past 70 years. Life tables have been prepared for males and for females for each decennial period from 1880 to 1950. The method of life table construction used is a derivation of the Reed-Merrell method, with the major difference being in the treatment of the age group 1–4 years by single years of age rather than as a group. The reason for this is that the rates of child mortality were so great in the early decades covered by the Reed-Merrell analysis as compared to present-day rates, that a consolidation of several years for our study would result in considerable loss of accuracy in total life expectancy.

The raw data needed a number of modifications and adjustments before mortality rates could be computed. Major adjustments were required in the population data for children under five years of age. A number of standard demographic techniques were employed to effect the necessary corrections both in the under-enumeration of population and the under-registration of births.[1] Further adjustments were required to effect comparability and also to supply missing pieces of information.

LIFE EXPECTANCY AT BIRTH

Total life expectancy at birth will be analyzed first. Table 1 presents the results for each decennial period.

TABLE 1

Expectation of Life at Birth and at One Year of Age, by Sex, City of Chicago: 1880-1950

	1880	1890	1900	1910	1920	1930	1940	1950
Males								
Age 0	38.0	37.0	46.2	45.8	54.3	56.6	61.5	64.8
Age 1	46.3	45.6	51.7	51.9	58.2	60.3	62.5	65.7
$\overset{0}{e}_1$ less $\overset{0}{e}_0$	8.3	8.6	5.5	6.1	3.9	3.7	1.0	0.9
Females								
Age 0	40.6	40.9	50.8	51.6	57.5	60.4	66.1	70.5
Age 1	46.8	47.9	55.4	56.8	60.4	62.6	66.8	71.0
$\overset{0}{e}_1$ less $\overset{0}{e}_0$	6.2	7.0	4.6	5.2	2.9	2.2	0.7	0.5

It should be noticed that the gain in average length of life was abut 27 years for males and about 30 years for females.

Very few series of life tables extend back as far as 1880. One such series exists for England and Wales. (Dublin, Lotka, and Spiegelman, '49, and United Nations Demographic Yearbook, '52). In 1880, life expectancy for males in England and Wales was 42.4 years, and in 1951 it was 65.8 years—a gain of 23.4 years; for females these figures were 45.8 years, 70.9 years, and 25.1 years, respectively. Thus, the net gain was not as much, but life expectancy was higher in England and Wales in 1880. Life expectancy in Chicago was very similar to that in England and Wales in 1950.

In addition to the net increase in life expectancy during the total 70-year period, it is also of interest to examine the extent of this increase by decades. Reference to table 1 will make clear that this increase was not linear in nature. It increased between 1890 and 1900, stayed at this level for a period (1910), and then increased to another level (1920).

[1] The birth registration could not be assumed to be complete until about 1925.

Length of life for males in 1890 was slightly less than in 1880. It was also true, but to a greater degree, in the states of New Jersey and Massachusetts, the only other areas of the United States for which life tables are available. The decreases in life expectancy in these early decades are characteristic of high mortality rates which are at the same time highly variable. As mortality rates decreased, they also became steadier.

In the period from 1890 to 1900, there was a 10-year increase in life expectancy, the greatest gain of any decennial period. Between 1900 and 1910 there was no increase, and a slight decline in length of life among males. By 1920 life expectancy was again increasing, and the 30-year period, 1920-1950, showed an increase in each decade.[2]

Differences in life expectancy at birth between males and females is most interesting. In 1880, females lived 2.6 years longer than males. This difference increased every year until 1910, when it stood at 5.8 years. In 1920 there was a sharp reduction in the gap, and it declined to 3.2 years. Again a steadily increasing difference prevailed until 1950 when it reached 5.7 years. The precise causes for this are not discernable here, but they are certainly worthy of further investigation.

LIFE EXPECTANCY AT SELECTED AGES

By subtracting the life expectancy at birth from the life expectancy at one year of age, the effects of mortality in the first year of life can be isolated (table 1). If infant mortality is excluded, the gain in life expectancy is not as spectacular although it is still of considerable magnitude. Life expectancy for males at one year of age has increased about 19 years since 1880, and for females about 24 years in the same period.

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[2] After 1920, Negroes represented a constantly increasing proportion of the population of Chicago. Mortality rates of Negroes have been higher than those of white persons residing in the same area. Thus, the life tables of 1880-1910 pertain to an almost wholly white population and the life tables of succeeding years are based partly on nonwhite persons. While it would have been possible to construct life tables for white persons only for these periods, it was not done here since the object was to show the life expectancy of the total population as affected by the sum of all social influences. If it could be shown that Negroes had a lower life expectancy because of biological differences, separate life tables for white persons would be justified. However, a study of racial and economic differentials in life expectancy in Chicago indicated that most of the difference in length of life between whites and Negroes is accounted for by economic differences (Mayer, '50).

Of the total gains of 27 years for males and 30 years for females, gains of 7 and 6 years, respectively, may be traced to the reduction in infant mortality. Infant mortality has declined in much the same manner as has total mortality. That is, infant mortality has declined each decade between 1880 and 1950 with the exception of 1900-1910, at which time it increased slightly.

With regard to differences between males and females in expectation of life at various ages we see in general that the gain in life expectancy for females has been greater than for males. In addition the increase in life expectancy for females has been steadier and less subject to fluctuations than that of males. Life expectancies of females have shown a net gain in each decade, except in 1880-1890 and 1920-1930, when small decreases were observed in the higher age groups. This is not as true for males, especially in the older age groups, and there has been an actual decline over the whole period for males above 55

TABLE 2

Net Change in Years of Life Expectancy at Various Ages for Decennial Periods, By Sex, City of Chicago: 1880-1950

AGE	1880-1890	1890-1900	1900-1910	1910-1920	1920-1930	1930-1940	1940-1950	1880-1950
Male								
0	—1.0	+9.2	—0.4	+8.5	+3.1	+4.1	+3.3	+26.8
1	—0.7	+6.1	+0.2	+6.3	+2.1	+2.2	+3.2	+19.4
5	—2.6	+3.9	0	+5.4	+1.0	+1.2	+3.0	+11.9
15	—4.1	+3.0	—0.4	+5.6	+0.5	+0.4	+2.8	+ 7.8
25	—3.4	+2.1	—0.8	+5.2	+0.4	—0.2	+2.4	+ 5.7
35	—3.5	+1.4	—1.0	+4.9	—0.1	—0.6	+1.9	+ 3.0
45	—2.8	+0.7	—0.8	+4.2	—1.6	+0.1	+1.3	+ 1.1
55	—2.3	+0.5	—0.6	+3.0	—1.3	—0.2	+0.5	— 0.4
65	—1.5	+0.1	—0.1	+1.8	—0.9	—0.1	+0.5	— 0.2
Female								
0	+0.3	+9.9	+0.8	+5.9	+2.9	+5.7	+4.4	+29.9
1	+1.1	+7.5	+1.4	+3.4	+2.2	+4.2	+4.2	+24.0
5	—0.8	+5.0	+1.2	+2.5	+1.1	+3.3	+4.0	+17.3
15	—1.8	+3.6	+1.0	+2.2	+0.9	+2.6	+3.7	+12.2
25	—1.4	+2.5	+0.7	+2.1	+0.4	+2.0	+3.4	+ 9.7
35	—1.2	+1.4	+0.4	+1.8	0	+1.6	+2.9	+ 6.9
45	—1.3	+1.0	+0.2	+1.7	—0.5	+1.3	+2.3	+ 4.7
55	—1.9	+1.5	+0.1	+1.1	—0.4	+1.0	+1.3	+ 2.7
65	—1.9	+1.5	+0.1	+0.7	—0.1	+0.5	+0.9	+ 1.7

years of age. This would seem to suggest that different or at least additional forces govern male mortality rates at a given time. Differences in occupation and social roles might account for this. However, the accuracy of the figures for ages above 55 years is questionable due to both errors of reporting and the method of life table construction employed here.

Summarizing these age differences as they appear in table 2 we can see that the bulk of the gain in life expectancy of both males and females is due to reduction in mortality rates among the young (0 to 15 years). Among males only 21% of the total gain in years of life can be attributed to reduction in mortality rates of men over 25 years of age.

Survivor Rates for the Period 1880-1950

While the expectation of life at various ages is a concise device for describing the mortality picture, another life table function—the number of survivors at various ages—portrays more graphically the social effects of mortality. Table 3 is based on the survivors of an initial cohort of 10,000 persons.

TABLE 3

Survivors at Selected Ages, by Sex, City of Chicago: 1880-1950

AGE	1880	1890	1900	1910	1920	1930	1940	1950
Male								
0	10,000	10,000	10,000	10,000	10,000	10,000	10,000	10,000
1	8,037	7,942	8,756	8,647	9,172	9,364	9,682	9,708
5	6,830	6,984	8,145	8,074	8,761	9,115	9,591	9,655
15	6,236	6,548	7,814	7,793	8,481	8,883	9,485	9,606
25	5,844	5,999	7,358	7,425	8,172	8,611	9,314	9,499
35	5,285	5,377	6,788	6,867	7,701	8,230	9,022	9,331
45	4,706	4,571	5,972	5,995	7,030	7,892	8,409	8,928
55	3,928	3,614	4,812	4,739	6,109	6,701	7,189	8,003
65	2,880	2,381	3,310	3,083	4,607	4,808	5,133	5,790
Female								
0	10,000	10,000	10,000	10,000	10,000	10,000	10,000	10,000
1	8,489	8,343	8,994	8,924	9,360	9,498	9,749	9,788
5	7,195	7,358	8,401	8,378	8,987	9,278	9,679	9,746
15	6,593	6,865	8,096	8,110	8,752	9,085	9,597	9,712
25	6,144	6,310	7,695	7,771	8,431	8,834	9,451	9,640
35	5,577	5,678	7,166	7,312	8,020	8,490	9,203	9,511
45	4,967	5,062	6,519	6,695	7,409	7,978	8,755	9,256
55	4,277	4,416	5,608	5,781	6,623	7,072	7,902	8,736
65	3,219	3,201	4,194	4,335	5,178	5,432	6,322	7,262

Of 10,000 infants born in 1880, almost 18% died during the first year of life. In the year 1950, of a cohort of 10,000 male births about the same number of persons survived to age 55 as survived to one year of age in 1880. In 1880, 58% of the males survived to 25, while about the same number survived to age 65 in 1950. Among females the changes have been even sharper. Almost the same number of females as attained one year of age in 1880 attained age 55 in 1950. In terms of the older age groups we see that more than twice the number of females attained age 65 in 1950 as did in 1880.

Table 3 demonstrates clearly that the past 70 years produced profound changes in the mortality picture in the city of Chicago. The social and psychological consequences of a far greater expectation of life on the family, the labor force, other social and cultural institutions, and social organizations in general have been far reaching.

The Generation Life Table

In addition to enabling us to analyze the mortality trends over a 70-year period, a decennial series of life tables makes possible the construction of a generation life table. Instead of using mortality rates as of a single point in time, the generation life table employs the observed mortality rates appropriate to a number of time points. For example, while the fixed mortality table for 1880 is based on observed death rates for all ages in the year 1880, the generation life table for 1880 is based on the 1880 mortality rates for persons under 10 years old,[3] the 1890 mortality rates for persons between 10 and 20, the 1900 mortality rates for persons between 20 and 30, and continues in this manner until the cohort reaches advanced age. Essentially, the fixed mortality life table is a description of a hypothetical population, since the mortality rates are as of a single point in time for all age groups. The generation life table may be applied to an actual population, and is therefore a more realistic picture of the mortality experience of a population group. Ideally then, the generation life table is a description of the mortality experience of an actual population living in a given locale. Actually, migration distorts the picture to some degree. Nevertheless, the generation life table comes closer to describing the mortality experience of a real population than does the fixed mortality life table.

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[3] This method of constructing a generation life table differs somewhat from that suggested by Dublin, Lotka and Spiegelman ('49) in that it is like a fixed mortality life table within each 10 year age grouping. If mortality rates are changing very rapidly this results in some loss of accuracy, especially in the first 10 years of life. However, the net effect on life expectancy at birth is small.

The 1880 generation life table can be said to apply to that cohort of persons born in Chicago in 1880, and living their life in that community. Comparison of this table with the fixed mortality life table for 1880 leads to the conclusion that the males have encountered an exceptionally severe mortality condition in their lifetime. The life expectancy at birth for males is the same (38.0 years) for the 1880 generation as it was for the 1880 fixed mortality table (38.0 years). Among females the life expectancy of the 1880 generation (42.9 years) exceeded its life expectancy at birth (1880) by 2.3 years.

A number of factors partially or wholly accounting for this situation might be suggested. Instead of the closed system as proposed above, there has in fact occurred a larger amount of immigration to this city. These immigrants have been predominantly male.[4] If the above tables could be constructed on the basis of nativity, different results might be found. Other possible factors might be: first, that this cohort was born too early to take advantage of any later advances in infant and childhood mortality reduction; second, that this cohort attained working age just at that time (1895-1900) when the hazards of industry and occupation were at their worst; and third, that this group reached the middle ages (55 years and over) just at the time that mortality rates for males increased slightly.

Rather than speculate on a situation for which evidence is lacking, it will be more fruitful to see if this situation applies to the cohort born in 1890. Accordingly generation life tables by sex were constructed for that cohort.

It is evident from these data (table 4) that the 1880 cohort did not fare nearly as well as the 1890 cohort in exceeding its life expectancy, as computed by the fixed mortality schedule as of the time time of their birth.[5] Further, in both cohorts, the females have exceeded their initial life expectancy to a greater degree than have the males. The cohort of 1900 fared somewhat differently during its collective life time. The life expectancy of males increased by 4.7 years and that of females by

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[4] In 1920 the sex ratio among the foreign born in Chicago was 116.

[5] Generation life tablest have been calculated for the 1890 Masachusetts cohorts, (Dublin, Lotka and Spiegelman, '49) and results were consistent with ours. In Massachusetts the 1890 male cohort outlived its life expectancy at birth by 4.2 years and females 5.8 years.

3.2 years. The greatest gains were made by males in the 5–34 year age group.

TABLE 4

Expectation of Life at Selected Ages for Generation Life Tables and Difference Between Generation and Fixed Mortality Life Tables for the Cohorts of 1880, 1890, and 1900, by Sex, City of Chicago

	1880		1890		1900[1]	
AGE	Gen. L.T.	Diff.	Gen. L. T.	Diff.	Gen. L. T.	Diff.
Male						
0	38.0	0	40.8	+3.8	50.9	+4.7
5	50.1	—0.1	52.9	+5.3	57.3	+5.8
15	44.2	—0.4	45.9	+5.4	49.4	+5.9
25	37.2	0	38.2	+4.4	41.2	+5.3
35	29.9	—0.7	30.9	+3.8	33.0	+4.5
45	23.5	—0.3	23.4	+2.4	24.2	+2.5
55	16.7	—0.8	16.6	+1.4	17.1	+1.4
65	11.3	—0.7	11.3	+0.8	11.7	+1.1
Female						
0	42.9	+2.3	45.6	+4.7	54.0	+3.2
5	53.1	+2.1	56.7	+6.5	59.0	+3.8
15	47.6	+2.3	49.9	+6.5	52.3	+5.0
25	40.7	+2.5	42.0	+5.2	44.2	+4.9
35	33.3	+1.7	34.3	+3.9	36.2	+4.4
45	25.9	+1.1	26.7	+3.2	28.2	+3.7
55	18.9	+0.9	19.5	+3.3	20.7	+2.9
65	13.1	+0.8	13.1	+2.7	13.8	+1.9

[1] As this cohort was only 50 years old in 1950, life expectancy for ages above 50 years was computed from 1950 death rates for persons 50 years of age and over. Our data show relatively little change has occurred in these rates in the past 70 years. However, it is more than possible that advances in medical science during the next 30 years will substantially change these rates downward and the cohort will outlive its life expectancy at birth by many more years than are shown here when it reaches its extreme life span about the year 2000. The same, but to a lesser degree, is true of the 1880 and 1890 cohorts.

SUMMARY

In summary, the analysis of both types of life tables clearly points to the progress made in medical science and health care during the past 70 years. An increase of 27 years for males and 30 years for females in life expectancy has not only been an effect but has been a cause of marked social change. When a "real" cohort of persons was traced through its collective lifetime it was seen that life expectancy at birth was an underestimate of the "real" average length of life of the groups. The cohorts of 1910, 1920, etc. are still too young to permit construction of a generation life table, but it is probable that they will exceed their life expectancy at birth by many years.

It is also probable that medical advances still to come, and their application to all persons in the population, will result in continually increasing life expectancy and that succeeding cohorts of babies will actually outlive their life expectancy at birth. By the time age specific mortality rates become so low that they are practically irreducible, science will be progressing on the problem of increasing the extreme life span.

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THE DISTRIBUTION PATTERN OF DENTAL CARIES

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IN collecting data on the epidemiology of dental caries various designations like D. M. F., *i. e.*, decayed, missing and filled (Klein *et al.*, '38 and '40, and Boyd, '43), and C. R. L., *i. e.*, carious, restored and lost (Barr, '49) have been used for both teeth and tooth surfaces. These indices are not always appropriate and sometimes may lead to confusion. At the time of examination two or more coalesced carious lesions may *appear* to involve two or more surfaces of a tooth and this destruction may, in reality, represent a one-surface lesion which has been left untreated and which has progressed so as to involve other tooth surfaces. Missing teeth may have a variety of reasons for their status. Many compound fillings do not indicate precisely which surface or surfaces were involved originally by the process of tooth necrosis. In addition to the foregoing, some operators place fillings in teeth which may not be defective at the time of operation, just to prevent them from becoming carious in the future.

Bodecker ('31) suggested that completely destroyed crowns of teeth, with only roots remaining, should count 5 points in the score of affected surfaces and lost teeth three points. Hollander and Dunning ('39) assumed in their study that each missing tooth represented three lesions. Klein and Palmer ('40) stated that if a one-surface involvement affects the pulp and necessitates extraction of a tooth, such severity could be considered as a 5-surface involvement by caries.

The author agrees with Wessels and Cheyne ('47) that while it is true that 5 surfaces of a tooth may be lost with the extraction of the tooth, this arbitrary consideration does not give information on the exact statistical status at the time of, or prior to the time of extraction. The reasoning of the investigators discussed earlier does not take into account the true biologic characteristics of dental caries. After considering the distortions which can be produced in the statistical results obtained by relying on the above-mentioned imperious assumption, it seemed desirable to conduct a statistical analysis of clinical data which did not necessitate the inclusion of any hypothetical material.

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PRESENT STUDY

Material

The present report deals with the patterns of proximal carious lesions in the permanent teeth of 140 white females between the ages of 18 and 21 years. All European ethnic strains commonly found in the United States were encountered in the group which, as a whole, represented the middle class population both socially and economically. All individuals examined were studying to become dental hygienists.

Method of Examination

A complete clinical examination of each dentition was carried out by the author, utilizing artificial illumination, a sharp probe and a mouth mirror. The examination and recording of findings were done with extreme care, taking about 15-20 minutes in each case. A complete set of both intraoral and bitewing X-rays was taken and the clinical recordings were carefully verified by comparing them with the radiographic findings. Each filling was taken to represent a carious lesion. Dental casts were available for each individual and they were analyzed for crowding and spacing of teeth and other characteristics of occlusion.

ANALYSIS OF DATA

For purposes of analysis only the mesial and distal surfaces were considered. Other surfaces were not included in the analysis, in view of the fact that prophylactic odontotomy is practiced by some dentists, so that in treating a proximal lesion the occlusal surface may also become involved. It was realized, also, that frequently it is necessary to extend a proximal cavity preparation to a non-proximal surface in order to secure access to the lesion and to obtain better mechanical anchorage for the filling. Third molars were not considered in this investigation since they were not clinically present in all the individuals studied, either because they were unemerged, or because they were absent due to agenesis (Nanda, '54) or early extraction.

All records of individuals with extraction of one or more teeth of the second dentition, or with teeth supporting jacket crowns, fixed bridges or partial dentures, were eliminated from the group before analysis.

A considerable number of young women with either natural spacing or crowding of the teeth was seen. As the relationship of spacing and crowding of the teeth to dental caries is still disputed, these subjects also had to be excluded from the analysis. Individuals who were completely free of dental caries were left out also, since they furnished no data of value in this type of research.

After the elimination of all of the above-described types of unsatisfactory basic clinical data, the remaining records were arranged in a serial order on the basis of an increasing total number of proximal lesions. The material pertaining to the maxillary and mandibular teeth was arranged separately. Eighty records of maxillary and 68 records of mandibular dentitions were available for detailed study. The number of records on maxillary and mandibular dentitions could not be kept equal. In order to obtain the maximum quantity of data for analysis, the data on the maxillary and mandibular dentitions were not taken necessarily from the records of the same individuals in all cases. It was assumed that the procedure followed would not cause any appreciable distortion in the findings.

FINDINGS

Bilateral Symmetry of Carious Lesions

Review of Literature. Leigh ('23) noticed the occurrence of dental caries to be bilaterally symmetrical, when groups of individuals were considered. Knutson and Klein ('38), Brekhus ('28), Cheyne and Drain ('40) noted a high degree of similarity between tooth mortality figures for the two sides of the oral cavity. Leigh ('41, p. 157) reported on the bilateral symmetry in the pattern of dental caries. Strusser and Dwyer ('41, p. 214), Hyatt ('41, p. 132) and Scott ('44) have pointed out that the phenomenon of bilaterality was found repeatedly in the material studied by them. Losee ('47) reported that the carious tooth surfaces occurred both in the abutting and bilaterally symmetrical pairs with greater frequency than if their distribution were determined by chance alone.

The results reported by the above-mentioned observers are based on the comparison of the total number of carious lesions in various morphologic groups of teeth. Study of data in this manner does not indicate the extent of bilaterality seen at the individual level. It is not clear, from the reports just discussed, with what degree of reliability the presence of a lesion in a particular surface of a given tooth can be predicted by examining its antimere.

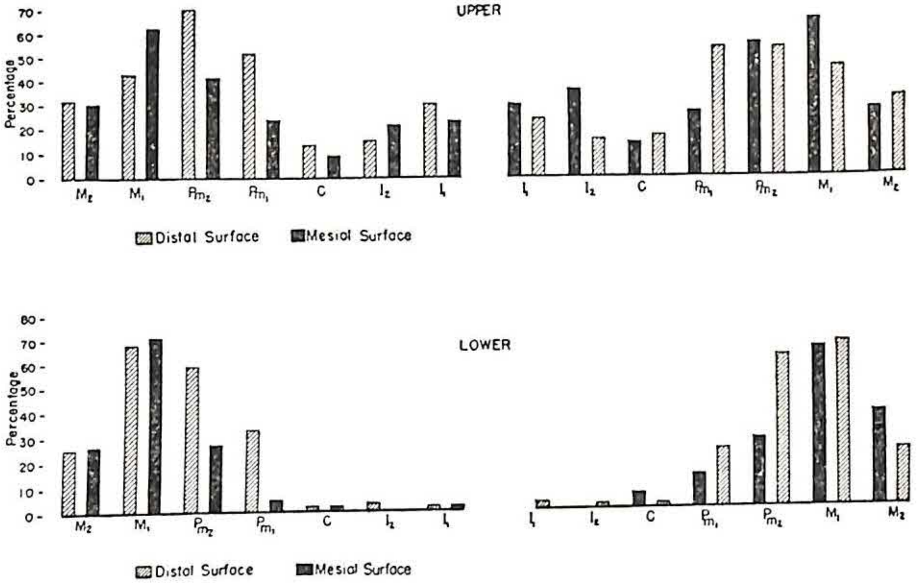

FIG. 1. The regional distribution of the caries in the proximal surfaces of permanent teeth, studied collectively in 80 maxillary and 68 mandibular dentitions. Each bar shows the percentage of carious surfaces in the sample. The chart has been arranged so that the right side of the mouth is represented on the left side of the chart and vice versa.

Barr ('49), from his study of over 500 individuals, reported a marked association between the occurrences of proximal surface caries in the teeth of the right and the left sides, studied collectively, but this high degree of bilateral symmetry was seldom encountered in individual cases.

The data were studied both collectively and individually. Figure 1 shows the "geographic" distribution of these lesions and also the collective degrees of bilaterality which exist in the group of individuals examined. The distribution and the bilaterality of the carious defects found in the proximal surfaces of the teeth examined are given in tables 1 and 2.

To determine whether the many instances of bilaterally occurring lesions in the corresponding proximal surfaces of antimeres could be due to chance, the Chi-square test[1] (with Sheppard's correction) was applied. As would be expected with the limited quantity of data at hand, the Chi-square values obtained differ considerably for the different morphologic classes of teeth (see tables 1 and 2). They could not be computed at all for the mandibular incisors and canines, as the number of carious surfaces observed in these teeth was too small for the purpose.

TABLE 1

Bilaterality of Caries in the Proximal Surfaces of 80 Maxillary Tooth Pairs

		CARIES	CARIES PRESENT ON				
TOOTH	SURFACE	ABSENT ON BOTH SIDES	LEFT SIDE ONLY	RIGHT SIDE ONLY	BOTH SIDES	χ^2	P
I_2	m	52	6	4	18	35.47	<.01
	d	48	8	13	11	7.57	<.01
I_2	m	47	12	4	17	22.14	<.01
	d	62	6	6	6	10.52	<.01
C	m	66	7	3	4	7.87	<.01
	d	62	7	5	6	10.55	<.01
Pm_1	m	47	14	12	7	0.80	N.S.*
	d	24	15	13	28	6.08	<.01
Pm_2	m	23	24	12	21	0.78	N.S.*
	d	16	8	21	35	4.63	.03
M_1	m	17	14	10	39	8.61	<.01
	d	29	17	14	20	2.49	N.S.**
M_2	m	45	11	13	11	2.23	N.S.**
	d	46	9	8	17	18.58	<.01

* Not significant between limits of .30 and .50.
** Not significant between limits of .10 and .20.

It is clear, also, on mathematical grounds that will not be discussed in this paper that the Chi-square test is of increasingly more limited usefulness the more the proportion of carious surfaces departs from 50% of the total number of the tooth surfaces under consideration. This means, in practice, that many of the Chi-squares obtained for molars and premolars should be interpreted with caution, and that the P values for these teeth should not be taken at full face value.

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[1] For the method of computation of the Chi-square values, the reader is referred to any of the numerous standard texts on statistics.

TABLE 2

Bilaterality of Caries in the Proximal Surfaces of 68 Mandibular Tooth Pairs

TOOTH	SURFACE	CARIES ABSENT ON BOTH SIDES	CARIES PRESENT ON			χ^2	P
			LEFT SIDE ONLY	RIGHT SIDE ONLY	BOTH SIDES		
I_1	m	67	0	1	0	—	—
	d	67	0	1	0	—	—
I_2	m	68	0	0	0	—	—
	d	66	0	1	1	—	—
C	m	64	3	0	1	—	—
	d	67	0	0	1	—	—
Pm_1	m	56	9	3	0	—	—
	d	37	9	15	7	0.63	N.S.*
Pm_2	m	42	8	7	11	11.37	<.01
	d	21	7	6	34	22.10	<.01
M_1	m	12	8	10	38	8.00	<.01
	d	11	11	10	36	4.30	.03
M_2	m	35	15	6	12	4.20	.04
	d	48	3	4	13	31.50	<.01

χ^2 cannot be computed where one cell is zero.

* Not significant between the limits of .30 and .50.

The reader should not draw the conclusion that it was felt that the Chi-square test, as employed here, gives an exhaustive or complete analytical account of bilateral symmetry in the clinical material studied. About all that can be said regarding P values tabulated in tables 1 and 2 is that, in general, they discredit any arbitrary hypothesis of no connection between the caries proneness of corresponding surfaces of right and left teeth. Probability values of less than 0.01 in particular suggest that the bilateral associations shown by the raw data are not explainable on the basis of pure chance alone.

The Occurrence of Caries in Adjacent Proximal Surfaces of the Teeth

The possible effect of a pathologic process in one proximal surface upon an adjacent proximal surface was also studied from the data available. The results obtained are shown in tables 3 and 4. The Chi-square test could be applied to only some of the data for contacting surfaces, because the total number of the defects in many such surfaces was too small for statistical analysis. Those surfaces for which caries figures were studied showed a high correlation between the occurrence of lesions in adjacent proximal surfaces, though the values of Chi-square varied considerably for different pairs of contacting surfaces. The tables have been presented in full, even for the teeth for which the Chi-squares could not be computed, as the recorded observations may be of interest to other students of the problem.

The data analyzed indicate a definite tendency to development of lesions in contacting proximal surfaces of adjoining teeth. Although no percentage values for distributions are shown in tables 3 and 4, it is evident that, in general, the proportion of lesions in *both* contacting surfaces is equal to, or more than, the proportion of lesions in only one of two such surfaces. This generalization cannot be made for the teeth which have an overwhelming preponderance of sound adjacent proximal surfaces. Moreover, it does not hold for the distal and mesial surfaces of mandibular M_1 and M_2, respectively, which seem to be remarkably dissimilar in their susceptibility to dental disease (see table 4, bottom line).

The data presented in tables 3 and 4 indicate the relative independence of many contacting teeth as regards their susceptibility to similar local environmental agents of tooth destruction and tooth preservation. The few small P values obtained do not rule out the probability of high degrees of constitutional individuality of many neighboring teeth in some dentitions.

The Distribution of Extracted Teeth

In this study care was taken to eliminate from the material analyzed all records that dealt with dentitions with either agenesis of teeth or loss of teeth due to accidents. Also, dentitions with teeth extracted for failure to emerge or for orthodontic reasons were not included. Following the processing of original data in the manner just described, the dental records of 100 individuals were available for the study of distribution pattern of teeth extracted due to caries.

TABLE 3

Occurrence of Caries in Adjacent Proximal Surfaces of 80 Maxillary Tooth Pairs

APPROXIMATING SURFACES				CARIES ABSENT FROM BOTH SURFACES	CARIES PRESENT IN			χ^2	P
TOOTH	SURFACE	SURFACE	TOOTH		FIRST SURFACE ONLY	SECOND SURFACE ONLY	BOTH SURFACES		
I_1	mesial	mesial	I_1	52	4	6	18	34.47	$<.01$
I_1	distal	mesial	I_2	63	2	7	8	—	—
I_2	distal	mesial	C	75	0	3	2	—	—
C	distal	mesial	Pm_1	73	2	3	2	—	—
Pm_1	distal	mesial	Pm_2	46	9	6	19	24.2	$<.01$
Pm_2	distal	mesial	M_1	33	11	11	25	14.06	$<.01$
M_1	distal	mesial	M_2	56	12	4	8	10.6	$<.01$

TABLE 4

Incidence of Caries in Adjacent Proximal Surfaces of 68 Mandibular Tooth Pairs

APPROXIMATING SURFACES				CARIES ABSENT FROM BOTH SURFACES	CARIES PRESENT IN			χ^2	P
TOOTH	SURFACE	SURFACE	TOOTH		FIRST SURFACE ONLY	SECOND SURFACE ONLY	BOTH SURFACES		
I_1	mesial	mesial	I_1	67	1	0	0	—	—
I_1	distal	mesial	I_2	67	1	0	0	—	—
I_2	distal	mesial	C	65	2	1	0	—	—
C	distal	mesial	Pm_1	64	1	3	0	—	—
Pm_1	distal	mesial	Pm_2	56	1	6	5	—	—
Pm_2	distal	mesial	M_1	21	8	12	27	10.0	$<.01$
M_1	distal	mesial	M_2	28	27	3	10	2.21	N.S.*

* Not significant between the limits of 0.10 and 0.20.

Excluding the third molars, 160 teeth were found to have been extracted from the afore-mentioned 100 dentitions: 71 of them were maxillary and 89 were mandibular teeth. The distribution of extracted teeth is shown in figure 2. Each bar indicates the percentage of extracted teeth in the group under study. The first molars in both jaws accounted for the majority of such teeth. Of the total of 160 extracted teeth, 104 were first molars. The mandibular first molars had the highest frequency of extraction, maxillary first molars were second in rank and the second molars in both jaws the third.

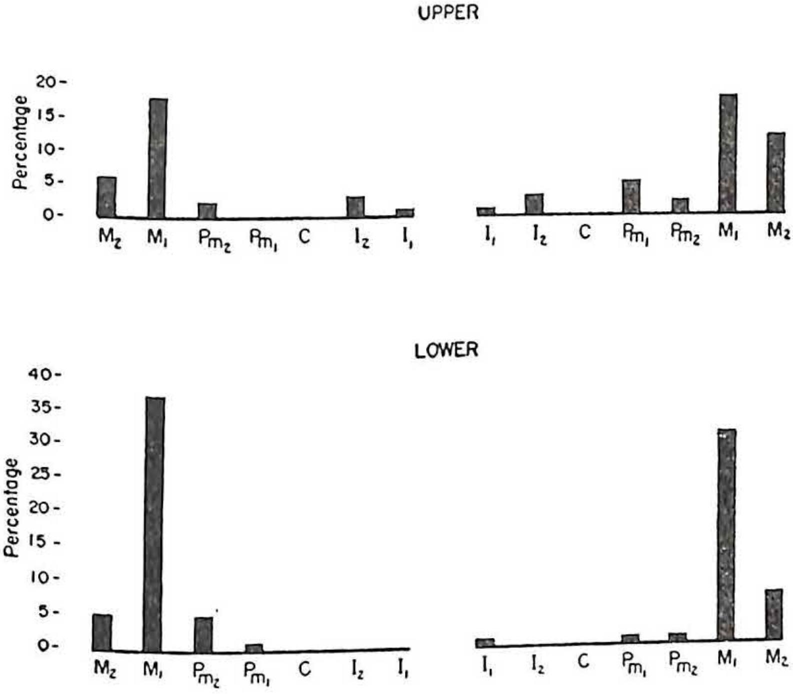

FIG. 2. The regional distribution of teeth extracted due to caries in 100 females. Each bar shows the percentage loss of a particular tooth in the group. The chart has been arranged so that the right side of the mouth is represented on the left side of the chart and vice versa.

A great deal of bilateral symmetry was again noticed in the pattern of extracted teeth, considered in terms of the total group, but this symmetry was not frequently encountered at the individual level.

Origin and Spread of Carious Lesions

(1) *Maxillary Teeth.* For the purposes of this study, the individual records for the maxillary and mandibular dentitions were arranged separately, in the order of the aggregate number of lesions in the arches. From the series of 80 maxillary dentitions, the 32 records, which indicated the presence of from one to 7 carious or filled surfaces in all, were singled out. The figures for a given proximal surface of the right and left teeth were arbitrarily added together and a table showing the aggregate number of lesions in the mesial and distal surfaces of each of the 7 teeth, first incisor to the second molar, was obtained. This was done in order to find out which proximal surface of a given maxillary tooth, considered collectively, is the most susceptible to dental caries or the most likely to be affected first. The proportion of defective proximal surfaces, out of the total of 64 proximal surfaces of a particular kind in this group of 32 maxillary dentitions, is expressed in the form of percentages in figure 3 for each of the two surfaces of each of the 7 classes of teeth. It is clear from this figure that the distal surface of the maxillary second premolar is the most frequently affected among the 32 individuals chosen as having relatively fewest proximal lesions in a total group of 140 young women examined.

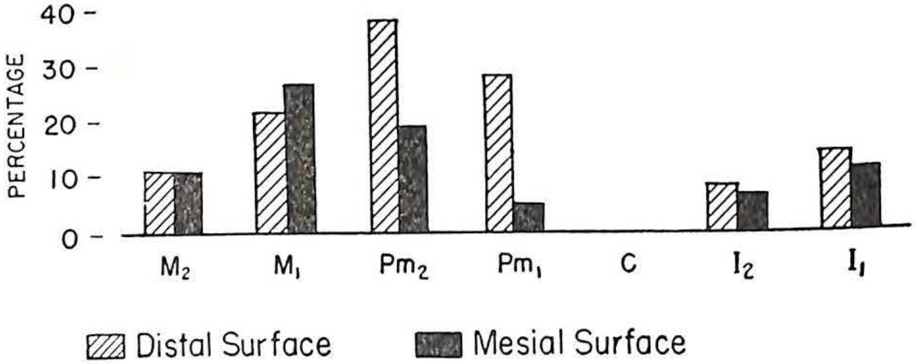

FIG. 3. The percentages of affected mesial and distal surfaces in the maxillary teeth of 32 individuals with a total of one to 7 defective proximal surfaces per individual maxillary arch. The figures for the right and left sides have been added together.

To determine further which surfaces are relatively most susceptible to dental caries, the data on the 32 maxillary dentitions mentioned above were divided into subgroups on the basis of different aggregate levels of proximal surface involvement. This was done to prevent any misinterpretation of results which might be produced by considering the data in larger groups. The calculation of percentages for each subgroup was carried out in the manner utilized for computing the percentages of affected surfaces in the entire group of 32. Figure 4 reveals that the distal surface of the maxillary second premolar is the most frequently affected surface in the group of 8 individuals with only one or two proximal lesions each. This was found to be true at all levels of proximal surface involvement, as shown in figure 5. Both the mesial and distal surfaces of the canines were seen to be the most immune to dental caries in this group. In general, the distal surfaces, both collectively and individually, showed a higher susceptibility to dental decay than the mesial surfaces of the affected teeth.

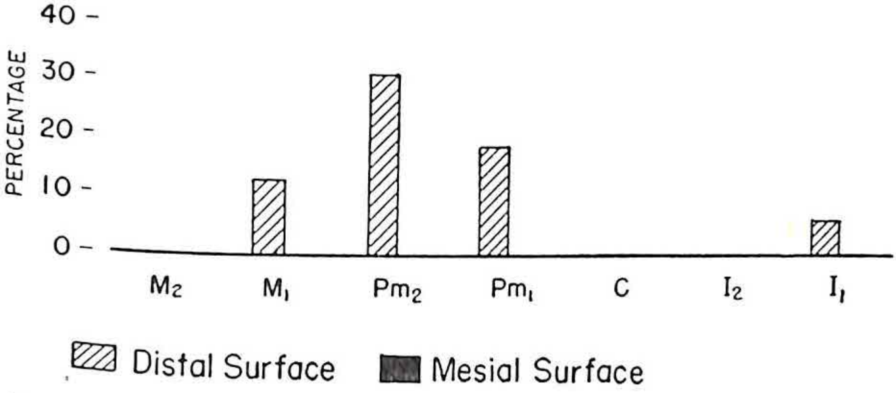

Fig. 4. The percentages of carious proximal surfaces in 8 individuals with 1 or 2 proximal lesions each in the maxillary arch. The distal surface of the maxillary second premolar is most often involved, while no mesial surfaces were defective in this group with the smallest number of proximal lesions.

From the records on maxillary teeth, arranged in order of increasing number of carious lesions, the 10 individuals with the smallest number of intact proximal surfaces were selected for further study. The number of sound proximal surfaces ranged from one to 10 per maxillary arch in this group. An attempt was made to find out what surface or surfaces were most immune to dental caries in a group which had been selected on the basis of a high susceptibility to the development of proximal lesions.

The data for the right and left sides of the mouth were arbitrarily combined, as in previous analyses. The percentages of sound surfaces,

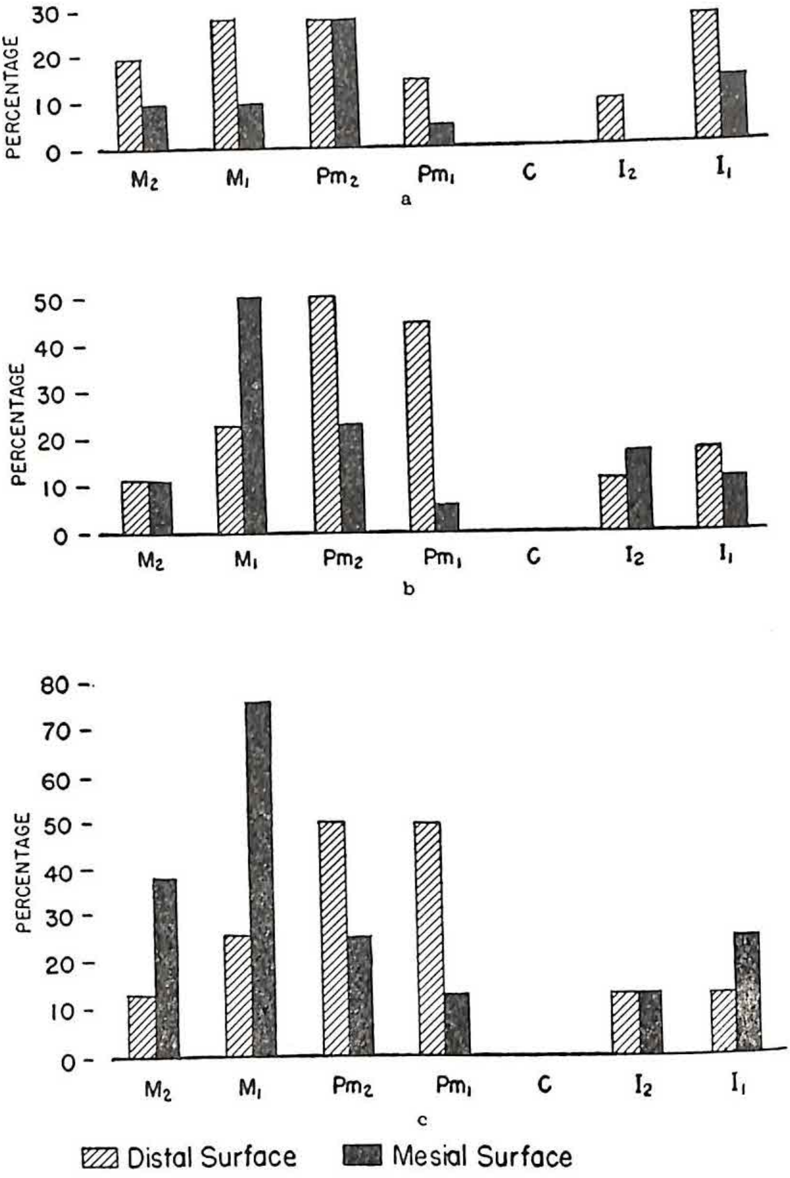

FIG. 5. Percentages of mesial and distal carious surfaces in the maxillary teeth of 24 individuals with different, relatively low degrees of carious involvement.

a. Eleven maxillary arches with three or 4 necrotic proximal surfaces each.

b. Nine maxillary arches with 5 or 6 necrotic proximal surfaces each.

c. Four maxillary arches with 7 necrotic proximal surfaces each.

considered collectively, are shown in figure 6, and for different levels of total involvement in figure 7. It can be seen that, among all the proximal surfaces, the mesial surface of the maxillary first premolar has the greatest immunity to dental caries. Next in rank are the distal surfaces of the central and lateral incisors and next to them the mesial surface of the canine. Even in this group the surface least immune to dental caries is the distal surface of the maxillary second premolar.

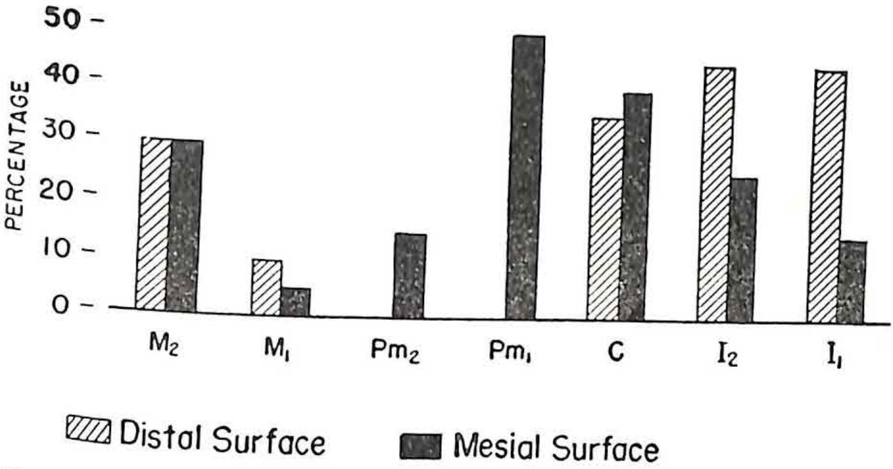

FIG. 6. The percentages of sound mesial and distal surfaces in the maxillary teeth of 10 individuals with one to 10 sound proximal surfaces per individual maxillary arch.

The present data indicate that the distal surface of the maxillary second premolar has the greatest chance to become diseased early and that it is the most caries susceptible surface in the maxillary permanent dentition, while the mesial surface of the first premolar is the most immune to dental caries among all the maxillary proximal surfaces.

(2) *Mandibular Teeth.* The data on the 68 mandibular dentitions, arranged in rank order of increasing number of proximal lesions, were treated in the same way as the data on the maxillary teeth. Twenty-five individuals with one to 5 proximal surface lesions each were considered first. As shown in figures 8 and 9, the mesial surface of the first molar was found to be the most frequently affected proximal surface, while the distal surface of the second premolar was next in order of susceptibilty. Both the mesial and distal surfaces of the first molar

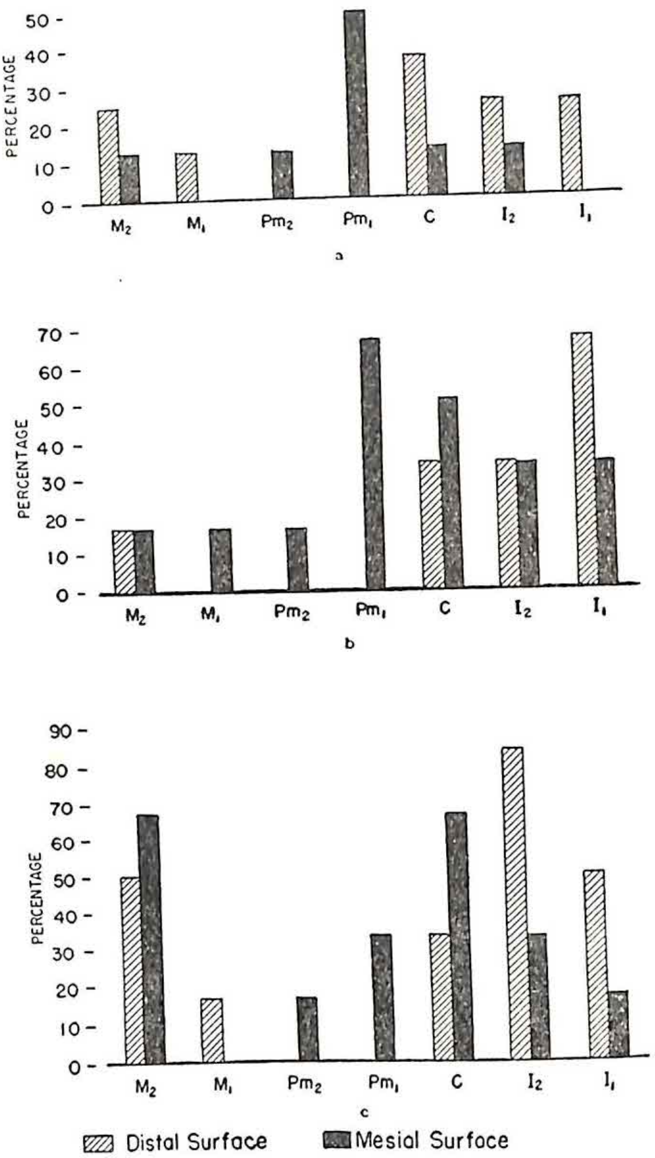

FIG. 7. The percentages of sound maxillary proximal surfaces in three subgroups of the 10 individuals with the highest relative susceptibility to proximal caries.

a. Four maxillary arches with one to 6 sound proximal surfaces each.
b. Three maxillary arches with 7 to 9 sound proximal surfaces each.
c. Three maxillary arches with 10 sound proximal surfaces each.

had the greatest tendency to early involvement at all levels of pathologic spread in the proximal surfaces. The mesial and distal surfaces of the canine, and of the central incisor, and the mesial surface of the second incisor showed the greatest immunity to dental caries in this group.

For the study of surface or surfaces with the greatest immunity to dental caries, in a group of the most susceptible individuals, 13 mandibular dentitions with 12 to 17 sound surfaces each were taken. The percentages derived are shown collectively in figure 10, and at various levels of spread of caries in figure 11. It is seen that the mesial surfaces of central and lateral incisors had the highest immunity to proximal caries. The least immune surfaces were the distal surface of the second premolar and the mesial surface of the first molar.

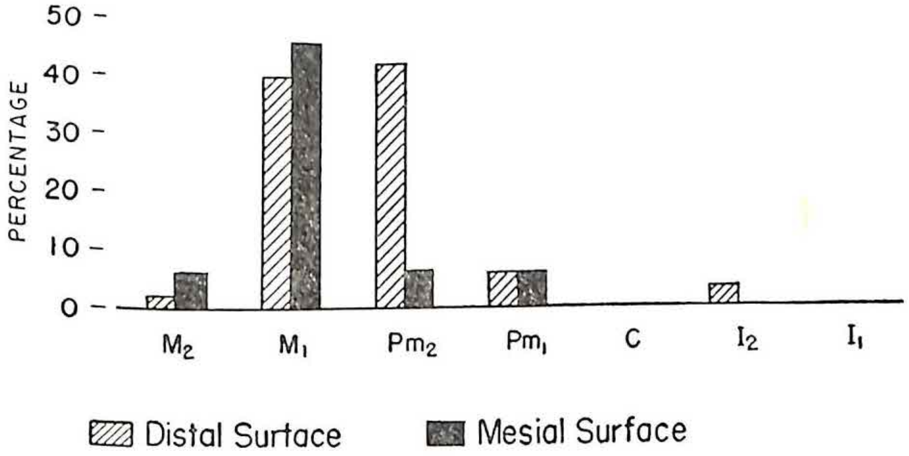

FIG. 8. The percentages of affected mesial and distal surfaces in the mandibular teeth of 25 individuals with a total of one to 5 defective proximal surfaces per individual mandibular arch. The figures for the right and left sides have been added together.

The mode of spread of carious lesions is evident from figures 3 and 8. The process of proximal caries involvement seems to start from the mesial surface of the first molar and the distal surface of second premolar. The frequency with which proximal surfaces of other teeth are involved seems to depend primarily on the susceptibility of the individual. The collective distribution pattern of proximal lesions in the different morphologic classes of teeth is shown in figure 1.

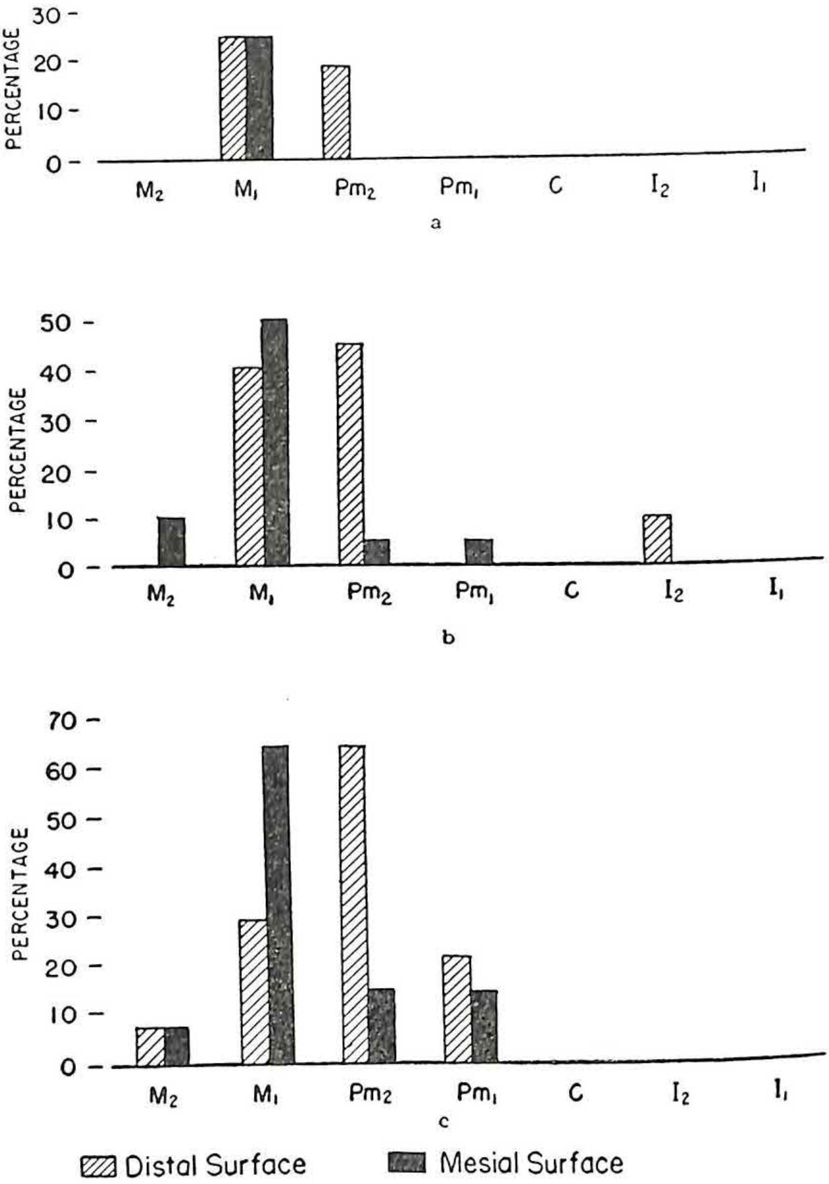

FIG. 9. The spread of carious lesions in the proximal surfaces of the mandibular teeth in three subgroups of the 25 individuals with a low susceptibility to proximal caries. Each bar shows what percentage of a particular proximal surface is affected in a given subgroup.

a. Eight mandibular arches with one or two necrotic proximal surfaces each.
b. Ten mandibular arches with three or 4 necrotic proximal surfaces each.
c. Seven mandibular arches with 5 necrotic proximal surfaces each.

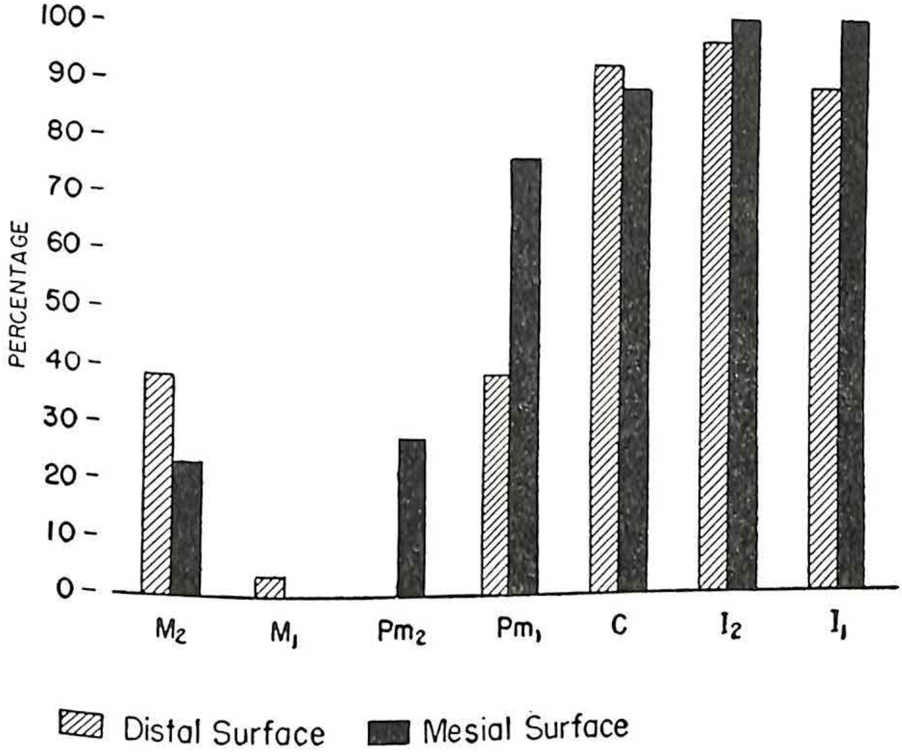

FIG. 10. The percentage distribution of the caries-free proximal surfaces in the mandibular teeth of 13 individuals with a high susceptibility to proximal caries. Each mandibular arch has a total of 12 to 17 sound proximal surfaces.

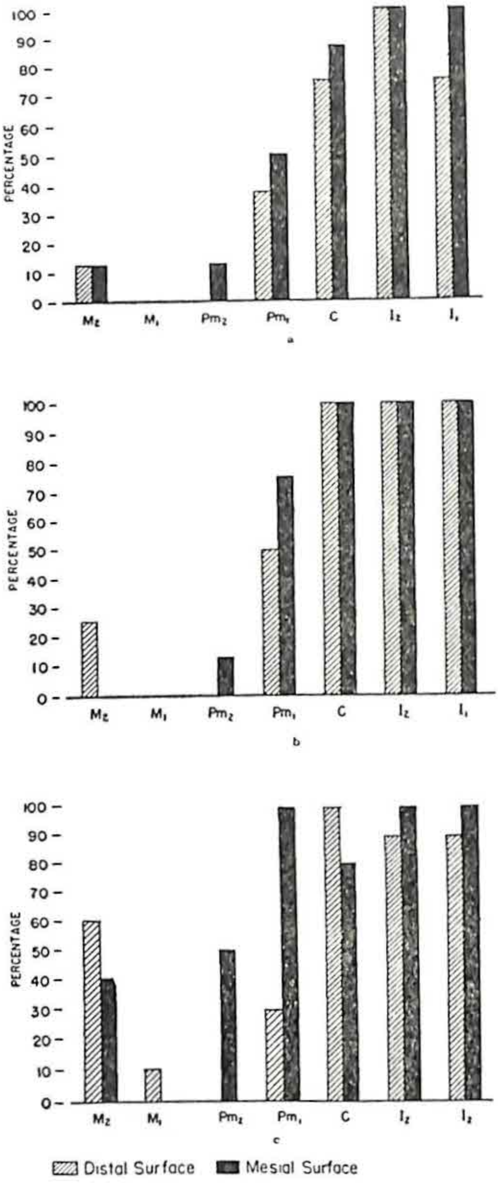

FIG. 11. The percentage distribution of sound proximal surfaces in three subgroups of the 13 individuals with a high susceptibility to proximal caries.

a. Four mandibular arches with 12 to 14 sound proximal surfaces each.
b. Four mandibular arches with 15 or 16 sound surfaces each.
c. Five mandibular arches with 17 sound proximal surfaces each.

SUMMARY

1. The pattern of spread of proximal carious lesions in 140 young, white, American women has been discussed.
2. Statistical tests in both individuals and groups indicate that the occurrence of carious lesions in corresponding proximal surfaces of antimeres—the phenomenon of bilaterality—is not fully explainable on the basis of mere chance and that, likewise, their occurrence in adjacent proximal surfaces cannot be attributed to accident in all instances.
3. The susceptibility of each tooth and each proximal surface varies greatly in different individuals.
4. There is a definite tendency toward a more or less specific regional distribution of lesions.
5. The first molars constitute the bulk of the extracted teeth. Mandibular first molars are more often extracted than maxillary first molars.
6. The usual point of origin of proximal dental caries is the distal surface of a second premolar or the mesial surface of a first molar, in either arch. The subsequent spread of lesions is determined chiefly by the individual susceptibility of each tooth and proximal surface, and usually follows a characteristic tautologous pattern.
7. The last proximal surfaces to succumb are, ordinarily, the mesial surface of the maxillary first premolar and the mesial surfaces of the mandibular central and lateral incisors.
8. No relationship between the length of time a tooth has been in the oral cavity and its susceptibility to dental caries was found.

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BOOK REVIEWS

Connective Tissue in Health and Disease. Edited by G. ASBOE HANSEN. 321 pp. Ejnar Munksgaard, Copenhagen, 1954. Dan. kr. 50.

This book represents a thorough resumé of the subject of connective tissue. The information is up-to-date, reviewed by investigators active in the field, and the bibliography includes nearly all of the significant papers dealing with connective tissue.

The list of authors include workers from Denmark, Sweden, Italy, Finland, England and the United States so that a broad viewpoint is presented. The entire subject is thoroughly and interestingly evaluated from the basic sciences dealing with structure, physical characteristics and chemistry, progressing through metabolism and biological behavior to a discussion of the clinical aspects and pathology of connective tissue. The book portrays the dynamic nature of the research that has been focused on connective tissue and the multiplicity of the lines of approach.

The connective tissue is comprised of collagen, reticulin, elastin and ground substance, with their associated fibroblasts, macrophages, mast cells, and minerals. The connective tissue fills the spaces between the cells through which everything going to and much of the material coming from the cells must pass. It transmits the energy of muscular contraction through a reticular sheath surrounding each fiber and thence to fascia, tendon and bone. In the case of the dermis supporting the epidermis, the connective tissue encases the body in a protective covering. The structure is specialized, depending upon function such as the transmission of light by the transparent cornea. Even the vitreous and aqueous humors and synovial fluid are considered to be connective tissue. Not only is it associated with many of the physiological functions of the body, but also it plays a major role in many pathologic processes and participates in repair following injury. With the realization of these facts, it is easy to understand the reason for the complex nature of the subject. Considering the connective tissue field as a whole, it encompasses perhaps every normal and abnormal bodily function, directly or indirectly.

Since the connective tissue in general is metabolically quite inert, it has in the past been neglected as an object of investigation. However, when ACTH and cortisone were shown to relieve so dramatically the symptoms of rheumatoid arthritis, a chronic connective-tissue disease, the entire thinking in medical practice and scientific investigation changed. For the first time it was widely realized that the inert, unchangeable connective tissue could be altered and that it was a fertile field of medical investigation. Greatly contributing to the aroused interest in the connective tissue were the new techniques being perfected—X-ray diffraction, electron-microscopy, protein chemistry and histochemical techniques. It was inevitable that this broad, unexplored subject with methods of attack at hand and general interest stimulated by the almost miraculous effect of the adrenal cortical hormones would attract an increasing number of investigators.

This book does not attempt to draw conclusions but presents and evaluates the facts in the field of connective tissue as they are now known. Such articles as the Histochemistry of Connective Tissue by J. F. H. McManus, the Chemistry of the Ground Substances of Connective Tissue by Karl Meyer, Hormonal Influences on Connective Tissue by Kurt Iversen and many others should be of interest to the general biologist. The information is presented in a way both refreshing and constructive. No biologist should miss it and the editor should be congratulated.

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Needed Research in Health and Medical Care: A Bio-social Approach. By CECIL G. SHEPS AND EUGENE H. TAYLOR. ix + 216 pp. University of North Carolina Press, Chapel Hill, N. C. 1954. $5.00.

Our understanding and control of disease has gained much in the past by incorporating the skills and knowledge of scientists who have not been initially and primarily concerned with medical or biological problems. The growth and contributions of biochemistry and biophysics are good examples of how fruitful the process of focusing the attention of men trained in other sciences on medical problems can be. This little book is the result of a deliberate attempt, conceived by the senior author, to encourage the social scientist to take an active interest in the problems of medicine. In September, 1952 the Rockefeller Foundation sponsored a conference at Chapel Hill to which were invited representatives of both the medical and the social sciences. The present publication is in effect the authors' personal report of this conference.

The book contains a carefully considered statement on the purpose of "biosocial" research, defines some of the areas that might be fruitfully examined, discusses problems in methodology, and presents certain organizational problems that arise in interdisciplinary work. In addition, there are three pre-seminar statements in the appendix which deserve study.

The material presented provides a good illustration of some of the difficulties of trying to obtain some understanding between scientists whose backgrounds are widely separated. The social scientists who attended the conference appeared to have had some common ground and understanding with the medical administrators and health educators, somewhat less with epidemiologists, and very little with the physiologists and internists interested in the etiology of major chronic diseases. Similar comments could be made about the comprehension of the medical personnel present regarding the tools and productive capacities of the social scientists. It is clear that there must be a good deal of education of both medical and social scientists before effective collaboration at the reasearch level (particularly when problems of the etiology of disease is concerned) can be achieved. Ways and means of obtaining such education are discussed under special organization problems.

This monograph is a "must" for anyone who wishes to work in the area of the relationships between social factors and the disease processes and should be required reading for those who are interested in the knotty problems of multi-disciplinary research.

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Bone: An Introduction to the Physiology of Skeletal Tissue. By FRANKLIN C. MCLEAN AND MARSHALL R. URIST. xii + 182 pp. University of Chicago Press, Chicago. 1955. $6.00.

The interests of the earlier physical anthropologists were mainly antiquarian and descriptive, with a strong emphasis on skulls. At about the point where lengths and breadths of bones and crude statistical seriations failed to yield anything very new or startling, osteometry, like other traditional procedures in gross anatomy, had passed its prime. It is fortunate indeed that the present century has seen so much productive work on *ossification*, as opposed to osteometry, so that now a respectable body of knowledge is available on the formation, maintenance and dissolution of the hard tissues. Rather than halting at the surfaces of dead bones, the new bone biologists have continued into the interiors of osseous tissues and treated them as living systems rather than as inert relics.

In the past decade, two syntheses of bone biology have been especially noteworthy: *On Bone and Bones,* by Weinmann and Sicher, and the newer work by McLean and Urist which is reviewed here. The former is the result of collaboration by a pathologist and an atomist, and the latter is by a physiologist and an orthopedist. In comparing the two works, it is surprising how little overlapping of subject-matter can be found. Weinmann and Sicher deal far more extensively with histology, bone diseases, and the growth of specific bones.

On the other hand, McLean and Urist hardly mention specific bones. Instead, they begin with morphology and histogenesis, and continue into a fascinating section on mineralization. The publication of this work is especially timely now, when agreement is rapidly being attained on the relationships between the crystalline lattice of bone and its constituents. Other important topics include the dynamics of calcification, enzyme action, resorption, endocrine agents, metabolism, tissue culture, transplants, the healing of fractures, and diseases of bone. A high proportion of the data came from the authors' own publications in nearly every field discussed in the book.

In some cases the bibliographies of the chapters are too sketchy to support all of the authors' statements. But a few forays outside of these bibliographies convinced this reviewer that the text must have been written with great care, attention to detail, and economy of words.

By virtue of this brevity, McLean and Urist hardly touch upon the morphogenesis of particular bones. A real need exists for an equally modern treatment of this field, but it would be difficult to find a team as good as these two authors to write it.

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Wirksame Faktoren im Leben des Menschen. (Operative Factors in the Life of Man) By OTMAR FRHR. VON VERSCHUER. Weisbaden, Franz Steiner Verlag. 1954. 288 pp. including 105 illust.

A longitudinal study of 159 pairs of twins of the same sex is summarized in this book. The twins were all studied by the author, first in 1924-5, later in 1926-7, 1936, and finally in 1950. For the purposes of this study, 150 pairs are considered (100 identical, 50 non-identical). The average age in 1924-25 was 16 years; in 1950, it was 40 years.

The focus of the discussion is on the relative changes which had occurred over the years rather than on the similarity or dissimilarity of the twins themselves. Of the 150 pairs, 18 had died by 1950; of 34 pairs, one of the pair had died. A high percentage (22%) of males were dead, largely because of World War II. Excluding those killed during the war, the author finds no corroboration of the folk-belief that when one twin dies, the other must follow him into the grave very soon. No regularity or significant difference between identical and non-identical twins is discernible, and all survivors lived a number of years after the death of the other twin.

Consideration of the causes of death (apart from war, of course), reveals that there is no correlation for either kind of twins. The ratio of likeness and unlikeness of cause of death is 9:69 for identical twins, and 4:32 for non-identical. This leads the author to conclude that it is impossible to separate out the hereditary and the environmental factors in disease, and impossible to predict the cause of death. Perhaps we should note that any sub-groups according to disease, such as cancer, heart disease, tuberculosis, and the like, are extremely small.

An interesting finding is the significant number of female twins, one of which is childless. Our author suggests that this might indicate disturbances in the development of the female sexual organs of the pair during pre-natal life.

There is extreme constancy in the bodily development of the twins, over the years. Far and away the most changeable measurement is body weight; then, in order, chest circumference, biacromial diameter, stature. In these latter, and indeed in all skeletal measurements, if the twins were orignally different, they keep about the same order of difference when older. The author emphasizes the constancy of the original body-type. As the subjects were, on the average, post-pubertal when first investigated, his results are not surprising. It is, however, unfortunate, that he does not evaluate them according to any available constitutional norms.

In the psychic area, great constancy is also manifested. One exception seems to be the emotional tone (*Stimmungslage*); changes are caused by purely environmental occurrences. The author admits that the psychic categories are not as well defined as the physical aspects. Nevertheless he maintains that psychic constancy is quite as marked as the physical.

A number of illustrations of the twins, predominantly of their heads, are presented. There is a complete table of the absolute measurements of the subjects, together with their ages, eye color and hair color. This reviewer would have appreciated a few indices as well.

In general, this book is strongly on the side of heredity rather than environment, except for physical diseases.

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The Distribution of the Human Blood Groups. By A. E. MOURANT. xvii + 438 pp. Charles C. Thomas, Springfield, Ill. 1954. $8.75.

More than one student of human biology has had cause to remark that the increase in the quantity and complexity of our knowledge concerning human blood groups has become so great that to keep up with it has become a full-time job. Thanks to the labors of Dr. Mourant this is no longer so, for in one authoritative, comprehensive, and readable volume he has made available all that the reader is likely to want to know concerning the distribution of the human blood groups.

In addition to the blood groups the author discusses such genetical characters of anthropological value as eye and hair color, PTC, thalassaemia, the sickle-cell trait, color-blindness, and the ability to smell hydrocyanic acid. A fair number of other traits—for example, internal canthic hairs, the palmaris longus muscle (the palmaris brevis is also of great genetic value), tongue furling, and tongue rolling—are not considered.

In separate chapters the author takes up the distribution of the blood groups among the peoples of the world, migrant and hybrid populations, and the blood groups of animals and their relation to those of man. There are excellent chapters on the blood grouping of bone and tissue specimens, the collection, preservation and transport of samples for blood grouping, and methods of gene frequency calculations for all the blood groups. A chapter devoted to recent discoveries deals with the new antigens. In a most useful chapter entitled "An attempt at synthesis" Dr. Mourant brings together what he regards as the most significant facts, and tries to show how they may be related to one another; he also helpfully speculates on how the blood groups may have arisen and by what stages they may have reached their present complex distribution.

There is an admirable bibliography of 1716 references, 9 maps showing the distribution of the blood groups throughout the world, 40 invaluable blood group frequency tables, a topological index to the bibliography, a zoological index to the bibliography, and a general index.

Reading this volume makes it clear that from being among the poorest genetically known animals man has, in the course of a few years, become one of the genetically best known; the "best," however, is here poor enough, for there remains a tremendous amount of rewarding work yet to be done. Meanwhile we owe a debt of deep gratitude to Dr. Mourant for this stimulating and helpful volume, a volume which is likely to remain the standard work on the subject for many years to come.

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Human Development. By JOHN P. ZUBEK AND PATRICIA ANNE SOLBERG. vii + 476 pp. McGraw-Hill Book Co., New York. 1954. $6.00.

The authors describe the book as "a survey of the extensive physical and psychological changes that occur through the course of life from conception through birth, adolescence and maturity to old age." This description in the introduction, like the statement in the preface that "the general orientation is physiological," is quite justified. For both casual perusal and careful reading reveal *Human Development* to be exactly as advertised. Take three parts psychological development, one part physical development, indicate wherever possible what is known about the genetics of behavior and hormone-behavior relationships, and the results will approximate the substance of this work.

Because this is a textbook, intended for students in child development or human development (wherever such a course is taught), its suitability as a text merits consideration. The book is well written. The many subsections are clearly set off by bold-faced captions, so that the student knows what he is reading about. The graphs and charts (and there are over 100 of them) are not only well chosen but for the most part carefully redrawn for maximum intelligibility. Brief summaries of the works cited or key quotations are given, thus avoiding the dangling citation so commonly found in elementary texts. And the 30 pages of references constitute an excellent working bibliography for advanced students desiring further knowledge of human development.

The first hundred pages on neural, glandular and physical development summarize a very broad range of findings, and include examples of the more commonly used measures of growth and development. Here, however, the authors' commendable desire to make the information interesting, occasionally leads them into the pitfalls of the popularizers. For example, in discussing the postnatal growth of the nose, they announce that "it . . . matures earlier than other body parts, thus temporarily distorting facial proportions" (p. 111). On the next page they nearly repeat in toto the familiar misconception that the child cannot walk until his bones have become calcified! Bone growth further troubles them as on page 119 where the number of ossification centers and the number of bones are confused. Minor errors, such as attributing the *somatotype* to Kretschmer (p. 117) and listing Krogman as a pioneer in the use of x-rays on *animals* (p. 120) are of less serious consequence. But sections on bone weight, muscle composition and muscle strength show that Zubek and Solberg have tried hard to assemble a complete account of physical development.

The final chapter on personality development may seem inadequate to many of us. Culture and personality, for example, are reviewed in only 11 lines, including the bare admission that "many personality traits may be culturally determined" (p. 410). The influence of parents similarly is dismissed in a short and uninformative paragraph (p. 409). Toilet-training, breast-feeding, swaddling, authoritarianism, parental indulgence, overprotection—all of these topics are conspicuous by their absence.

Yet these omissions are consistent with the whole plan of the book. For Zubek and Solberg rely heavily on experimental studies and on carefully conceived and adequately reported research. Much of the published information on the determinants of personality, it must be admitted, fails to come up to these standards. Furthermore, throughout *Human Development* the stress is on the genetic, endocrine, and morphological aspects of development so that the authors need not be censured for their intentional disemphasis on the cultural and situational determinants of the individual personality.

Without question *Human Development* is a commendable and useful elementary text. It should prove valuable to the teacher as well as the student since it affords the psychologist a rapid review of human biology and the biologist an acquaintance with a substantial section of human psychology.

STANLEY M. GARN

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The Functional Body Measurements of School Age Children. By W. EDGAR MARTIN. vi + 90 pp. National School Service Institute, Chicago. 1954. $1.00.

During most of the history of our industrial civilization, little scientific effort was made to produce goods or equipment in conformity with the physical dimensions of the human body. More recently, the fields of design and anthropometry have begun to interdigitate. Since a majority of professional anthropometrists occupy academic rather than industrial or governmental positions, the initiation of this fruitful alliance has come largely from non-anthropologists.

Since the onset of World War II, the dire necessity of fitting airmen effectively into cramped battle stations in aircraft and human faces snugly into gas or oxygen masks, has brought anthropometrists actively into military research. Projects such as the design of prosthetic appliances, clothing, shoes, or work spaces in vehicles are now the active concern of several physical anthropologists. These specialists can be found in a number of military installations, particularly the Quartermaster Research and Development Laboratories, Natick, Mass., the Army Chemical Center at Edgewood, Maryland, and the Wright-Patterson Air Force Base, Dayton, Ohio. Comparable work has been done abroad on a more limited scale.

In civil life, it is also apparent that seating and work spaces in many instances do not fit the American adult. A pioneer attempt to improve this situation was the anthropometric survey of seating, published in 1945 by the late Professor E. A. Hooton of Harvard University. This project was initiated by the Heywood-Wakefield Company of Gardner, Mass., in an effort to improve its products. This manufacturer has subsequently produced some excellent new seats by using Professor Hooton's data. More recently, a team at the Harvard School of Public Health, under Professor Ross McFarland, has studied the possible improvement of the dimensions of driver's compartments in American trucks.

One of the difficulties in designing work spaces or seats for American adults is that the mature population of this country is considerably heavier and slightly taller than its nineteenth century predecessors. As a result, antique furniture and the staircases and banisters in old houses are often too low, small or frail by present-day standards.

An even more conspicuous difference in size between Americans today and their ancestors is found in children. At a given age during the school years, an average American child is now considerably nearer his adult size and weight than was a corresponding child in the nineteenth century. This acceleration of growth often makes ordinary school furniture considerably more uncomfortable for a modern child than antique chairs and tables are for modern adults. Such cramping of school children in unsuitable furniture is the problem which stimulated Dr. Martin's study.

This report is based on a set of 55 measurements which were performed on 3,318 school children in southern Michigan in 1954. Some of these dimensions were those used by older anthropometrists, but others were made with the subject seated in an adjustable chair with a desk. Such realistic equipment has been a fundamental part of most modern seating surveys, including those of Professors Hooton and McFarland.

Dr. Martin's report is based on a survey planned and executed by the Laboratory of Physical Anthropoology of the University of Michigan, under the supervision of Professor Frederick P. Thieme. It was subsidized by the National School Service Institute, 27 East Monroe Street, Chicago. This group is an association of manufacturers of school furniture and supplies, and we can therefore expect them to make good use of the data.

The findings are presented in a simple and practical manner: some seriations are divided in accordance with grades in primary school, and others by chronological age from 5 years to maturity. All measurements are not only described fully in the text, but are illustrated above each statistical table on simple mannikin figures.

This report is a very valuable reference work, and can be understood easily by designers and other non-anthropologists. Among possible applications of the data which Dr. Martin mentions is the design of desks, laboratory and shop benches, and bookshelves. Some of the findings might also be applied to children's vehicles and dental chairs. We cannot be certain, however, whether future generations of American children will outgrow these specifications and have to be measured again.

In the meanwhile, here is a project whose execution shows a high standard of professional excellence. Both the text and the tables have been prepared with the clear intent of making the report widely useful. It should be in the hands of every manufacturer of children's vehicles and equipment. A copy is in my work space, and this report deserves to be easily accessible to every professional physical anthropologist.

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The Fossil Evidence for Human Evolution: An Introduction to the Study of Paleoanthropology. By W. E. LE GROS CLARK. x + 181 pp. University of Chicago Press, Chicago. 1954. $6.00.

If you were to discover a new fossil man, one of the first persons whose opinion you would seek would be W. E. Le Gros Clark, professor of anatomy at Oxford. He has studied, described and discussed many important skeletal remains, notably those of Proconsul and the Australopithecinae from Africa. The search for an author of a small book synthesizing what is known of fossil man for an audience of well-informed biologists (the purpose of the Scientist's Library series in biology and medicine, of which this work is the second title) virtually narrows to Le Gros Clark alone. Few, if any, others share his experience in the study of primate fossils, knowledge of biological principles, and mastery of methods.

If the reader knows enough of the subject to grasp the meaning, he will appreciate the terse descriptions, the wealth of pertinent details, the information about important discoveries of the last few years and the lucid statements of the author's own views on many of the still unsettled problems of interpretation.

Le Gros Clark applies principles of general evolution to his interpretation of human fossils. He mentions the possible roles of natural selection, mixture, random genetic drift and mutation. For instance, he says: "While the principle of irreversibility of evolution is perfectly sound in its general application, it is not legitimate to use it as an argument against ancestral relationships in reference to isolated characters which may have quite a simple basis and which are not obviously related to any marked degree of *functional* specialization. It is well known that some mutational processes may be reversible, for this has actually been demonstrated by genetic studies in the laboratory."

The dual approach to understanding human evolution through study of the fossils and analysis of the process is sound. But it is not quite achieved. It still remains for someone to write a self-sufficient text for the beginning student. Le Gros Clark gives much space to the definition of the scientific Latin names used in paleoanthropology. Whatever he gains in precision thereby is offset by two disadvantages: one, those previously unfamiliar with the terminology will find it difficult to follow the argument; and two, the emphasis on the formal taxonomy focuses attention on types and stages in such a way that the reader, despite the occasional mention of selective adaptation and genetics, easily loses sight of the *course* of human evolution.

One example may illustrate the problem. Together with most other students of fossil man, Le Gros Clark believes that modern man is less closely related to Western European Neanderthal man than to men known from remains of earlier pre-Würm age. However, Le Gros Clark alters the usual emphasis. He says: "On the basis of the morphological and chronological evidence at present available, the most reasonable (but still quite provisional) interpretation is that a primitive type of *H. sapiens* came into existence by the Middle Pleistocene, presumably from an earlier small-brained type represented by the *Pithecanthropus* stage of hominid evolution; that *H. neanderthalensis* arose as an aberrant (and, in some respects, a retrogressive) collateral line from pre-Mousterian or early Mousterian *H. sapiens*; and that the former species eventually became extinct."

In order to defend this taxonomy, in which he derives an extinct species from the surviving one, Le Gros Clark is obliged to define *H. neanderthalensis* narrowly and to emphasize certain features of the cranial base and the post-cranial skeleton which are preserved in only a few of the specimens. And since the creation of a third category would be of dubious value, he must then include within the species *H. sapiens* a wide range of variants including the specimens from Steinheim, Fontéchevade, Swanscombe, Ehringsdorf, Quinzano, Krapina, Saccopastore and Mount Carmel (in part). Some of these are Neanderthaloid in respect to large brow ridges, thick skulls, extremely taurodont teeth, small mastoid processes, poorly developed chins and some degree of platycephaly. One wonders why Le Gros Clark retains, and uses so much space to redefine, the specific name, *H. neanderthalensis,* for he says: "The recognition of a specific distinction on the basis of a fossil record which is by no means complete must, of course, involve an element of doubt. But the chronological evidence at least indicates rather strongly that *H. neanderthalensis* was a typical divergent radiation *in process of speciation,* even if this process *had not become quite complete in the genetic sense* by the time this group of Paleolithic man became extinct." (Italics are the reviewer's).

For the most part the interpretations now most widely held follow closely those of Le Gros Clark. He groups the fossil finds into a gradational series of types—modern *Homo sapiens,* Early Mouterian and Acheulian man, Pithecanthropus (including Pekin man and "Meganthropus"), Australopithecus, and Pliocene and Miocene anthropoid apes. He views such a series as chronological (with the possible exception of overlap of Pithecanthropus and Australopithecus) as well as morphological. Hence, although he thinks it unlikely that we have yet found the remains of our very ancestors, he presents this series as one of ancestral human stages.

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Free and Unequal: The Biological Basis of Individual Liberty. By ROGER J. WILLIAMS. xiii + 177 pp. University of Texas Press, Austin, Texas. 1953. $3.50.

Addressed primarily to the lay reader, this book is written in a popular and dramatized style, with no bibliography, footnotes, or any other formal citation of sources. The author presents essentially three points: (1) individuals differ widely; (2) these differences are derived largely from heredity; and (3) human variation should be taken into account in such matters as social and educational planning, group relations, and inter-personal contacts of daily living.

In developing the first point, Dr. Williams refers to individual differences in a wide variety of human characteristics, ranging from fingerprints and the chemical composition of urine to mathematical aptitude and artistic preferences. Well over half of the book is devoted to convincing the reader that people do differ—widely and in all traits. Some attention is likewise given to intra-individual variation and trait patterns. Thus the author reiterates the fact that people cannot be adequately classified in terms of a uni-dimensional measure such as an IQ, but should be compared in separate abilities. All of this is, of course, elementary knowledge to anyone who has taken a beginning course in psychology. It is undoubtedly desirable to present such information in a vivid and popularized manner for lay readers, as this book does. But it is regrettable that the book leaves one with the impression that such findings are new or even revolutionary. Nowhere is the reader told that during more than half a century psychologists have conducted many hundreds of studies on the nature, extent, and causes of individual differences.

The author's principal object in writing this book appears to be a plea for more consideration of individual differences in the practical business of everyday life. This, too, is a positive contribution and provides a number of thought-provoking ideas. The weakest part of the book, however, is the assumption of an hereditary basis for all the reported human differences. Moreover, it is apparent that the author considers this assumption fundamental to his thesis, although there is no reason why it should be so.

The treatment of heredity is confusing, replete with logical fallacies, and lacking in evidence. The confusion is caused by loose use of terms and poorly formulated questions. For example, the author writes, "We must not lose sight of inborn differences" (p. 23). Indeed we must not! Nor must we assume that such inborn differences are hereditary. Throughout the book, the author refers to the "newborn baby" as his symbol for heredity (*cf.*, *e.g.*, pp. 3, 7, 52). Nowhere is the reader given any inkling of the extensive research on the influence of prenatal environmental factors in the etiology of feeblemindedness or other behavioral and structural deviations in both human and infra-human organisms. In fact, the book strongly reenforces the popular error which identifies birth with the beginning of environmental influences.

Another misconception is that unless a characteristic can be attributed to deliberate training, it must be hereditary. Thus we are told that "there is a wide diversity of tastes which are not simply trained into people (as is often thought) but are based upon inborn differences of a profound and basic nature" (p. 80). Similarly, there is a recurring implication throughout the book that characteristics which are not hereditary can be changed at will at any time in the individual's life (*cf.*, *e.g.*, p. 140). Underlying much of the book is a gratuitous assumption that environmentally produced differences are superficial and impermanent and that they can be ignored for all practical purposes. This reflects a very limited and naive concept of environment, which is quite at variance with that found in modern biology and psychology.

In support of his contention that individual differences in aptitudes are hereditary, the author refers briefly to Thurstone's current research on homozygous and heterozygous twins. In so far as the pattern of aptitudes was more similar in the former, it is concluded that, "Every newborn baby has a distinctive and complex pattern of inborn mental capacities" (p. 52). No account is taken of the greater environmental similarity in the case of homozygous twins, in comparison both to heterozygous twins and to ordinary siblings, a point which has been repeatedly discussed and richly documented in the psychological literature. Similarly, the author cites many instances of the effect of hormone injection upon such behavior as nest-building and "peck order" hierarchy in various species. He does not, however, mention the equally impressive array of data regarding the influence of early experience upon the subsequent behavior of animals.*

Moreover, inherent in the author's reasoning is a confusion between physiological correlates of behavior and hereditary origin. The establishment of correlation between behavioral and physiological characteristics is a useful intermediate step in the study of hereditary factors. But it is by no means a demonstration of hereditary origin. The author advocates further research on physiological correlates of behavioral differences in human subjects (pp. 154-159). He fails to mention that such studies have so far yielded uniformly unpromising results. To be sure, recent developments in the author's own field of biochemistry, as well as in other sciences, may lead to more fruitful results. But we must guard against putting the conclusion before the data, as the author seems to have done. We must wait until future research has demonstrated physiological correlates of behavioral differences, which in turn are traced to heredity, before we can make the sweeping generalization that all important human differences result from heredity.

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* A recent survey of such data can be found in F. A. Beach and J. Jaynes. 1954. Effects of early experience upon the behavior of animals. *Psychol. Bull.*, *51*: 239-263.

In a chapter entitled "Races, People, Individuality," the author very properly calls attention to the wide individual differences found within each racial group and to the fact that no group can be rated as superior or inferior to another in *all* traits. To illustrate the latter point, however, he choses a weak and dubious example. Thus he suggests that Negroes as a group may excel whites in "capacity for personal loyalty and devotion, for emotional warmth, for religion, for music, for laughter and carefreeness" (p. 124). At least some of the traits included in this example fit into a familiar stereotype which is gradually being dislodged as a result of psychological research and social reform. All such trait differences, moreover, the author attributes to hereditary racial differences. He makes no mention of the mass of research which demonstrates the influence of cultural factors upon group differences. Nor does he seem to be aware of the fact that ethnic differences in aptitude patterns have been investigated. He categorically asserts that such groups have been compared only in general "intelligence" (p. 123).

The author's enthusiasm for the importance of "inborn" (hereditary?) individual differences sometimes involves him in queer logical tangles. Thus he writes, "Further, if we conclude that babies differ from each other in inconsequential ways, we are forced to accept another corollary: One baby could be substituted for another without substantial loss, and there is no consequential basis for regarding the life of any one child as distinctly valuable" (p. 11). Surely the fact of human variability is not the only—or even the major—argument against the wholesale slaughter of babies!

Finally, it should be noted that despite the author's predominant emphasis, the hereditary basis of behavioral differences is not, as he maintains, a radically new idea, calling for an "upheaval in basic thinking" (p. 165). On the contrary, it represents a return to an early viewpoint, away from which the behavioral sciences have been moving during the past 70 years, as the concept of environment has become broadened and refined and as pertinent data have been accumulating. It is unfortunate that the positive contributions of this book are so inextricably intertwined with fallacious and unfounded statements as to render its net value questionable.

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Symposium on Fatigue. Edited by W. F. FLOYD AND A. T. WELFORD. viii + 196 pp. H. K. Lewis and Co., Ltd., London, 1953. $4.00.

This publication represents the first volume of the Proceedings of the Ergonomics Research Society, a group founded in Great Britain in 1950 for the purpose of studying the relations between man and his work environment from the differing points of view of the anatomist, physiologist, psychologist, industrial physician, and engineer.

Symposium on Fatigue consists of twenty papers on the physiological and psychological aspects of fatigue, delivered before the Society in March, 1952. The lack of a specific definition of fatigue as well as the complexity of the whole concept can be noted throughout much of the report. Although the precise nature of fatigue is still imperfectly understood, its existence on a physiological basis is indisputable, as in the case of heavy muscular work where it can be related to the amount of exertion in terms of the number of calories expended by the human body. In his study of iron foundry workers, Christensen adds further significant criteria based on measurement of oxygen consumption, pulse rate, body temperature and metabolic rate.

Psychological fatigue, on the other hand, is demonstrated by tasks in which no heavy muscular effort is expended but in which fatigue can still be observed. The two factors usually associated with this type of fatigue are 1) a diminishing output and 2) an increasing effort. Sir Frederic Bartlett points out that both of these criteria are fairly rough and are quite susceptible to variation under many different conditions. As alternatives he substitutes the following as more characteristic of psychological fatigue: 1) an increasing irregularity of the successive items of a task, 2) the failure of the operator to take full advantage of all available information to guide him in his task, and 3) subjective symptoms such as pains and irritations. Of primary importance in this psychological approach to fatigue is the behavior of the organism as a whole. However, a deficiency in the knowledge of the specific physiological functioning of the central nervous system limits a fuller understanding of the problem. A further difficulty is that fatigue may often be confused with other phenomena such as boredom which at times produce similar manifestations.

The various approaches and the subject-matter presented in this report are sufficiently diverse to give a good coverage of current research into the meaning and causes of fatigue, psychological as well as physiological. The results of the original studies, as well as the discussions of previous work, should be of interest to all those concerned with any phase of human biology as well as to those concerned with problems of worker productivity in industry.

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Symposium on Human Factors in Equipment Design. Edited by W. F. FLOYD AND A. T. WELFORD. viii + 132 pp. H. K. Lewis and Co., Ltd., London. 1954. $4.00.

The second volume of the Proceedings of the Ergonomics Research Society is concerned with the application of knowledge of human capabilities and limitations, biological and psychological, to the most efficient design of all aspects of equipment and working areas. In this sense it is similar to what is often referred to as "human engineering." The current need for such a biological approach to equipment design is best exemplified by the following excerpts from one of the engineering contributors to the symposium.

"The machines of the engineer are becoming more and more complicated with a corresponding tendency to become more difficult to control. We can no longer afford to leave the human operator to get along as best he can. . . . It is evident that the problems we have to deal with have a strong biological flavor . . . and although our knowledge of the fundamental biological problems involved is far from complete, enough is known to justify the introduction of biological considerations as a distinctive contribution to machine design."

The 15 papers included in the symposium indicate clearly the range and type of contributions that can be made by the various biological and related sciences, varying as they do from recommendations for the design of chairs and tables, or discussions of human responses to design variations in visual displays, to statements of the physiological effects of climate or the circulatory disadvantages of a standing position for certain types of work.

Insofar as each paper presents the specialty of the individual researcher, it may be noted that the volume exhibits a certain lack of cohesiveness. However, the wide scope of the material certainly makes this somewhat unavoidable in such a symposium. This volume is a most worthwhile achievement when considered as a statement of some of the basic problems in the field, or as a survey of some of the data and of methods currently applied to problems of equipment design. To those unacquainted with human engineering, it can serve as an excellent introduction to some of the aspects of this relatively new field. It demonstrates beyond any doubt the importance of the contributions the biologist can make to the field of equipment design.

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Indian Health in Arizona. By B. S. KRAUS. ix + 164 pp. University of Arizona, Tucson, Arizona. 1954. $2.50.

This publication is based, principally, on a study of records of the Bureau of Indian Affairs hospitals on the Papago, Pima, San Carlos, Colorado River and Fort Apache reservations and the Phoenix Medical Center in Arizona for the fiscal year 1952-53. The Health Branch of the Phoenix Area Office contracted for this work in order to obtain a comprehensive analysis of health conditions and a description of customs of specific tribes which could be used to secure more ready acceptance of modern medicine on the part of Indian patients.

The report acquaints the reader with the location, geography, and population distribution of the Indians on these reservations. It also outlines the organizational pattern of the Bureau of Indian Affairs and the money budgeted for health. A brief but limited discussion of the preventive medicine program is presented. The tabulation on the use of clinic and hospital facilities was done with thoroughness and hence one can gain a reliable picture of the response of the Indian patient to health services. The difference in the validity of hospital records versus the clinic cards should be kept in mind in interpreting the statistics. The outpatient records were often made without adequate diagnostic facilities.

In spite of these limitations, the report should be noted as the first analysis of its kind of morbidity among Indian populations. It makes available much factual information on Indian health and health services in the Southwest, not previously available.

In recent years there has been a marked increase in the interest in "geographic medicine" and in the mode of life, including diet, as a factor in the development of so called "degenerative" diseases, such as coronary heart disease. The study of well selected groups of American Indians may contribute importantly to the clarification of this large and complex problem.

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BOOK NOTICES

EMOTIONS AND BODILY CHANGES. By Flanders Dunbar. xxii + 1192 pp. Columbia University Press, New York, 1954. $15.00. The fourth edition of a systematic survey of literature (1910-1953) on psychosomatic interrelationships. Orientation and methodology (180 pp.). Organs and organ systems (500 pp.). Therapeutic considerations and conclusions (70 pp.). Bibliography (330 pp.).

A TEXTBOOK OF PHYSIOLOGY. By John F. Fulton. xlii + 1275 pp. W. B. Saunders Co., Philadelphia, 1955. $13.50. Seventeenth edition of W. H. Howell's classical textbook. Nervous activity (240 pp.). Muscle (60 pp.). Sensory functions (200 pp.). Blood (60 pp.). Circulation (240 pp.). Respiration (80 pp.). Body fluids and kidney function (80 pp.). Digestion (70 pp.). Metabolism and nutrition (100 pp.). Endocrines (70 pp.). Reproduction (50 pp.).

THE VERTEBRATE BODY. By Alfred Sherwood Romer. viii + 644 pp. W. B. Saunders Co., Philadelphia, 1955. $7.00. Second edition of an introductory account of vertebrate morphology, with considerations of paleontological background, embryological history, histological structure and physiological function.

FAT METABOLISM: A symposium on the clinical and biochemical aspects of fat utilization in health and disease. Edited by Victor A. Najjar. viii + 185 pp. Johns Hopkins Press, Baltimore, 1954. $4.50. The aim of the symposium was to bring to the attention of the clinical investigator the pertinent new aspects of biochemistry and acquaint the biochemist with the clinical problems. Obesity was examined in reference to the multiplicity of causative factors (Jean Mayer, B. Childs, L. Wilkins) and clinical aspects of obesity in childhood were considered (H. H. Gordon).

THE MERCK INDEX. By Merck and Co., Inc. xiv + 1167 pp. Merck and Co., Inc., Rahway, N. J., 1952. $8.00. Sixth edition of the encyclopedic work on chemicals and drugs. There are 160 pp. of appendices, including different systems of measures and weights, and a table of radioactive isotopes and their uses in medical diagnosis and therapy. Seventh edition is scheduled for 1960.

STATISTICAL METHODS FOR THE BEHAVIORAL SCIENCES. By Allen L. Edwards. xvii + 542 pp. Rinehart and Co., Inc., New York, 1954. $6.50. Introductory textbook, with special reference to psychology, sociology and education. Aims to make the subject-matter accessible to students with a minimum amount of mathematical training. Incorporates methods of statistical analysis free from the assumption of normality of distribution. Devotes a chapter to significance tests for ranked data.

STUDIES ON THE NUTRITIVE VALUE OF BREAD AND ON THE EFFECT OF VARIATIONS IN THE EXTRACTION RATE OF FLOUR ON THE GROWTH OF UNDERNOURISHED CHILDREN. By E. M. Widdowson and R. A. McCance. viii + 137 pp. (Medical Research Council Special Report Series No. 287). Her Majesty's Stationery Office, London, 1954. $1.90. Children in two orphanages in western Germany were divided into groups and given, in addition to their German rations, as much as they wanted of one of several specially prepared breads made from flours of different extraction rates. Clinical status, biochemical data, radiographs of elbow and wrist, dental caries and tooth structure were used as criteria. Metabolic balances were also carried out. No significant differences in the nutritive value of the different breads could be detected.

ANATOMY OF THE HUMAN BODY. By Henry Gray. 1480 pp. Lea and Febiger, Philadelphia, 1954. $16.00. The appearance of the 26th edition, edited by C. M. Goss, documents the time-tested usefulness of this work concerned primarily with systematic anatomy. A 50 page chapter is devoted to embryology. Surface and topographic anatomy is presented briefly (60 pp.), Quantitative considerations are essentially absent.

STUDIES IN SCHIZOPHRENIA. By Robert G. Heath and 22 co-authors. xiv + 619 pp. Harvard University Press, Cambridge, Mass., 1954. $8.50. A progress report on research on mind-brain relationships, utilizing the techniques and concept of psychiatry, psychology, physiology, biochemistry, neurology, and neurosurgery. Theoretical framework for a multi-disciplinary approach to human behavior (50 pp.). Animal studies (60 pp.). Human studies (260 pp.). Discussions (50 pp.). Focus: Facilitation of cortical activity by electrical stimulation of the septohypothalamic region.

FOURTH ANNUAL REPORT ON STRESS. By Hans Selye and Gunnar Heuser. 749 pp. Acta, Inc., Montreal, 1954. $10.00. An indispensable guide to contemporary literatures on stress. General considerations, including the concept of stress, pathways of responses to stress, development of "adaptation syndrome," interpretation of "diseases of adaptation." Ten articles by guest authors. Systematic index to special physiology and pathology of stress. References (close to 5,000 items). Index of alphabetically arranged cross-references.

THE LANGUAGE OF SOCIAL RESEARCH. Edited by Paul F. Lazarsfeld and Morris Rosenberg. xiii + 590 pp. The Free Press, Glencoe, Ill., 1955. $6.75. A reader in the methodology of social research, it consists of reprints of papers, plus introductory and connecting text, grouped into 6 sections: Concepts and indices, Multivariate analysis, Analysis of change through time, Formal aspects of research on human groups, Empirical analysis of action, Toward a philosophy of the social sciences.

SUICIDE AND HOMICIDE. By Andrew F. Henry and James F. Short, Jr. 214 pp. The Free Press, Glencoe, Ill., 1954. $4.00. An attempt "to bring within a common framework knowledge and theories about the direction of expression of aggression which have developed independently within the disciplines of sociology, psychology and economics." Part I. Relation to business cycle (economic correlates). Part II. Sociological determinants. Part III. Psychological determinants of the choice between suicide and homicide. Appendices.

THE EVOLUTION AND GROWTH OF HUMAN BEHAVIOR. By Norman L. Munn. x + 525 pp. Houghton Mifflin Company, Chicago, 1955. $6.50. A comprehensive textbook bringing up-to-date the author's *Psychological Development* (1938). Coverage: Genetics (40 pp.), Phylogenetic psychology (unlearned behavior, evolution of intelligent behavior, 100 pp.), Ontogenetic psychology (prenatal behavior, sensory processes, motor development, symbolic processes, intelligence, emotional development, social behavior, personality from infancy to senescence, 350 pp.).

HUMAN ENGINEERING GUIDE FOR EQUIPMENT DESIGNERS. By Wesley E. Woodson. 245 pp. University of California Press, Berkeley, 1954. $3.50. To aid engineers by considering the characteristics and limitations of the human operator. The compilation of information was initiated by the Electronics Design and Development Division, Bureau of Ships. Topics: Equipment and work space, Vision, Audition, Body size, Movement, Body sensitivity, Orientation, Bibliography of design applications (16 pp.).

HANDBOOK OF HUMAN ENGINEERING DATA. 2nd edition. By Tufts College, Institute of Applied Experimental Psychology. 400 pp. Tufts College, Medford, Mass., 1952. Aims to "supply information on the practical limits of human performance and sensitivity, beyond which the man, as a control device for machines, begins to break down." Nine parts: Human engineering, Experimental techniques, Body dimensions, Space requirements in aircraft, Vision, Audition, Skin sensitivity and proprioception, Motor performance, Physiological conditions, Intelligence, Learning, and Glossary and Indices.

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BLOOD GROUPS, RACES AND PREHISTORY

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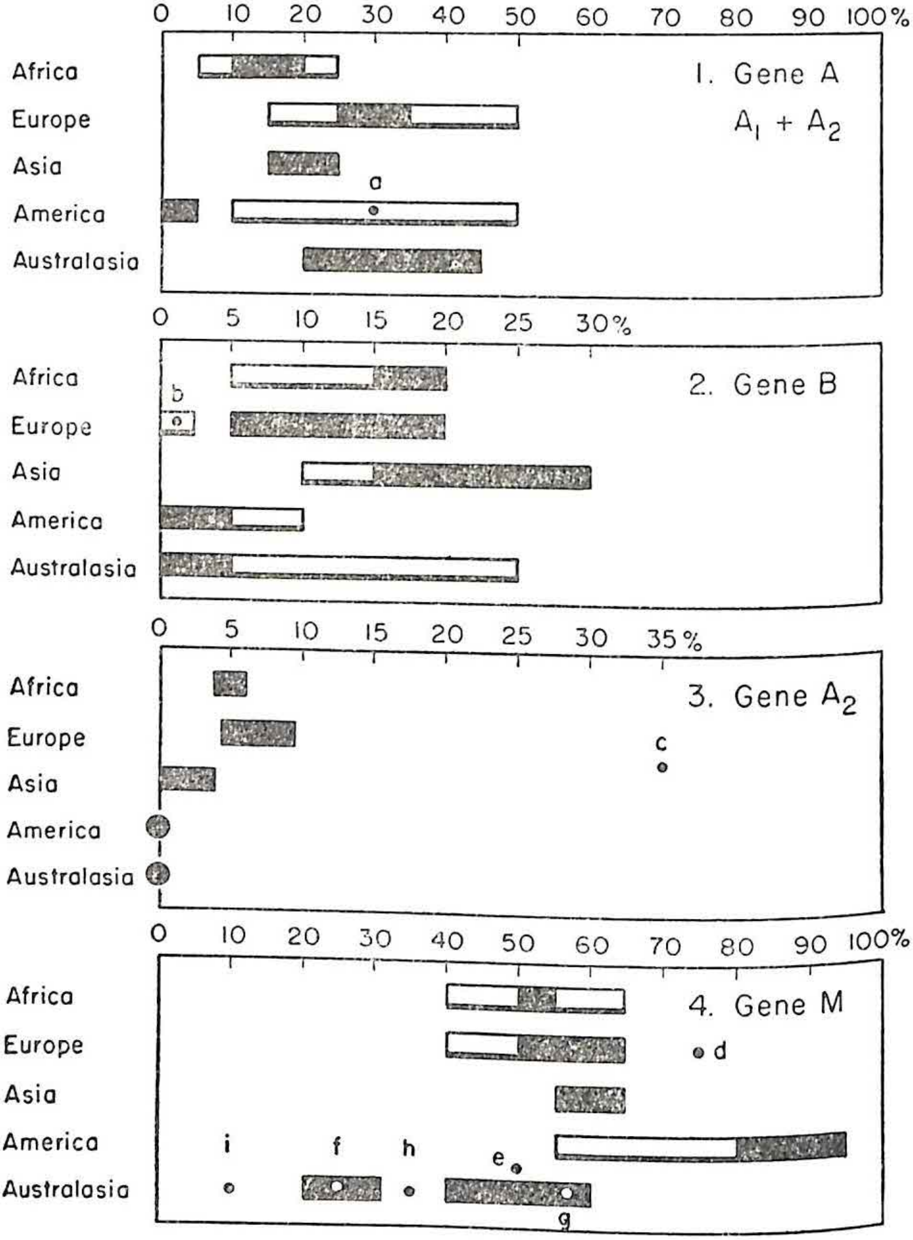
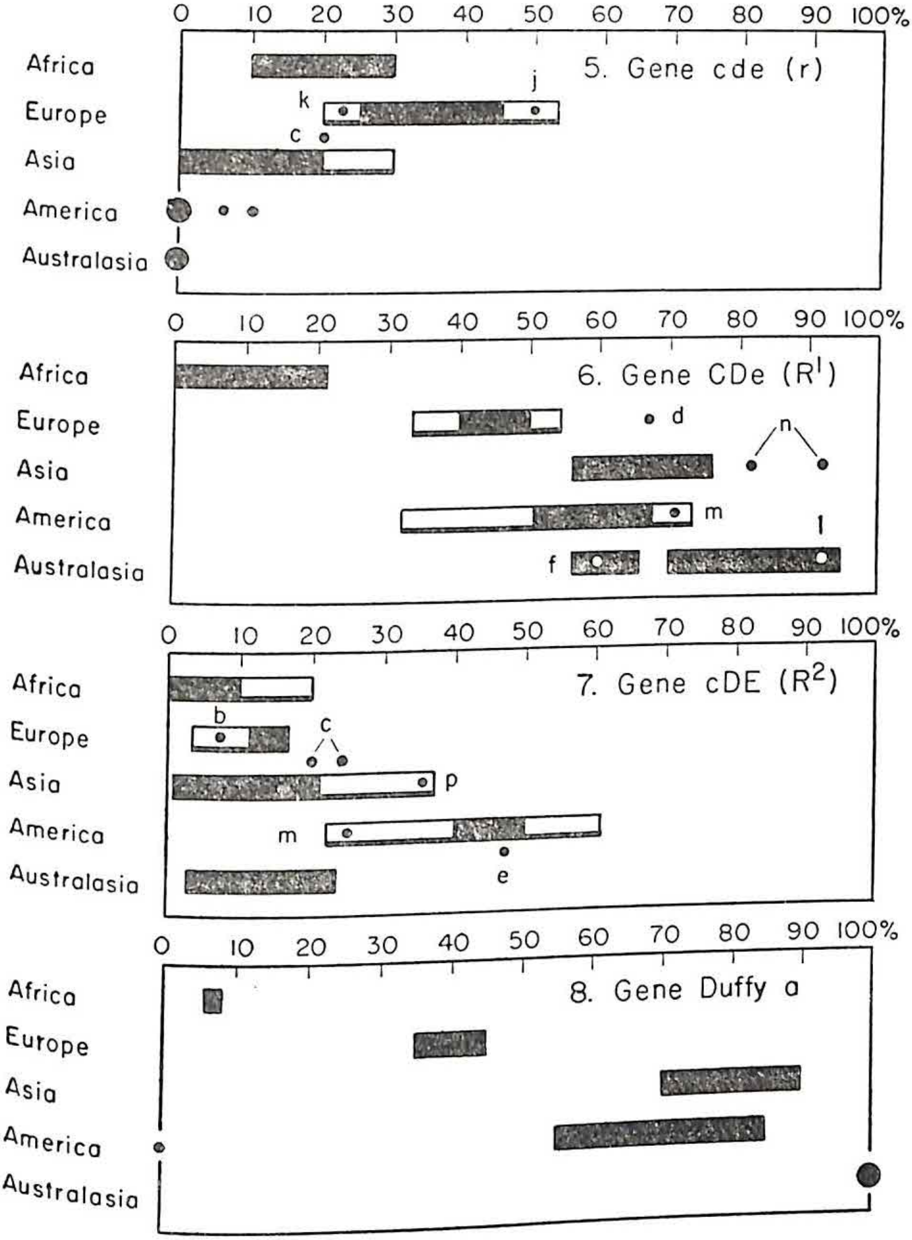
NOW that the vast material on blood group distribution over the world has been fully compiled by Mourant ('54) one may attempt to condense the outstanding data in a synoptic form. Short schemes as presented in this note necessarily disregard important details, but they should have didactic value and it will be seen that they lead, moreover, to interesting speculations.

The schemes have in the main been confined to the 5 great races: the Negroes; the Caucasoids, including Mediterraneans as far as the Sahara and the Caspian Sea as well as Russians as far as the Urals; the remaining Asiatics, most of them typical Mongoloids; the American Indians and the Eskimos; the Australasians comprising the Australian aborigines and the populations of Oceania (Indonesians, Melanesians, Micronesians, Polynesians). Eight blood genes, comparatively well explored or yielding strong racial differences are considered.

There are numerous serological characteristics of the Negroes, three of which are shown in the schemes. They have less *CDe* (R^1) gene (below 25%) and less *Duffy* (Fy^a) gene (below 10%) than the other



FIGS. 1-4. BLACK PORTION OF BAR: MOST COMMON PERCENTAGES.
a. North American Indians. b. Basques. c. Lapps. d. Sardinians. e. Maori. f. Australian natives. g. Indonesians. h. Micronesians. i. Melanesians.



FIGS. 5-8. BLACK PORTION OF BAR: MOST COMMON PERCENTAGES.
b. Basques. c. Lapps. d. Sardinians. e. Maori. f. Australian natives. j. Basques, Berbers, Irish. k. Sardinians, Greeks. l. Australasians. m. Eskimos. n. Andaman Islanders. p. Japanese.

races; and the A_2 values of Negroes as of whites are surpassed only by those of the Lapps. Whites, specially the Basques, are rich in *cde* (*r*); however, this peculiarity is less marked in the Mediterranean area. Besides, no gene is known which would separate the whites from all other races at once, although they differ from each race in particular by several gene frequencies. Asia shows high frequencies of B, R^1 and *Duffy*. Some genes pass through Asia in gradients: The moderate values of r in the Mediterranean area seem to proceed with small variations as far as India, vanishing beyond this almost completely; A_2 decreases in the same direction, whereas R^1 goes the opposite way. The American Indians maintain the record of *cDE* (R^2) and together with the Eskimos also of M, but A_2 and, virtually, r are absent in both. The Australasians share with them the lack of A_2 and r, and have much R^1 and *Duffy*, the latter gene reaching sometimes 100%. There are sharp subdivisions, however. Indonesians still exhibit the high Asiatic B and M values. Melanesia and Micronesia are characterized by minimum of M and maximum of R^1. Australian aborigines, if pure, seem to lack B (and S). The Polynesians approach Amerindians in M, R^2 and A.

These well known data may exemplify the use of such schemes in teaching. Yet to take another point, they disclose some suggestive convergences, based on which we will try, with all reserve, to interpret some results of serological anthropology in a more general manner.

What at first attracts our attention is the similarity of the "profiles" of A_2 and r on the one hand and of R^1 and *Duffy* on the other. Among the 5 races the Australasians have the lowest values of the first two genes, and the highest ones of the other two. This configuration is hardly due to chance alone, since the probability of one extreme value of any gene for one out of five races is .4, and for the 4 genes, 0.4^4, about 0.026. In fact, the probability is even smaller because those 4 gene frequencies are in Australasians not merely higher or lower than in the other races, but approach or reach 0 or 100%.

Other variables than chance must be invoked to account for the accumulation of extreme values in Australasians. It clearly cannot be the result of mixing with any of the 4 other races. One could imagine that selection acted on r, selection or mutation on A_2, fluctuation on R^1 and *Duffy*. Be this as it may, mechanisms would have worked slowly always. Hence the association of 4 extreme values, together with other pecularities, points to an ancient race which originated in Australasia or Eastern Asia: the protomongoloid race.

This race would have been fixed before the separation of the American Indians, related to it by the frequencies of all those 4 genes. Possibly the Amerindians brought their high *M* and R^2 frequencies along from Siberia where data are lacking. Their high O frequency has less weight, *ABO* frequencies being less stable.

The Negroes form the opposite extreme to the protomongoloid picture regarding R^1 and *Duffy*, and they have in addition unique values for *ABO* secretion, *P* and particularly *cDe* (R^0). The probability of chance combination of five extreme values would be but 0.4^5, nearly 0.01. No scheme has been drawn for R^0 because part of the observations is not quite clear before extended by D^u tests. At any rate the high R^0 frequency in Negroes is so striking a feature that it alone suggests that the race is very old: the protonegroid race.

Serological evidence for the two old races may be correlated to some concepts of palaeo-anthropology. It has been claimed by several authorities that the ancient Javan forms (*Pithecanthropus* and Solo Man) can be linked with Australian natives of today, as well as the Rhodesian Man with living South African populations. On the other hand, no link has been found in Europe between Neanderthal Man and the modern Europeans. If anthropological characters of living people are traceable back to so remote an ancestry, it is not unlikely that blood gene frequencies may be more stable than is generally supposed. In any case the idea deserves consideration.

Gene frequencies of Europe and Asia, in contrast to those of Australasia and Africa, are mostly not extreme but intermediate, showing that repeated intensive mixture occurred even in historic times. Thus the white race appears comparatively young, as suggested also by the *B*-gradient, the high value of *r* and the very low value of *CDE* (R^z).

Apart from Australasians and Negroes other smaller populations with exceptional blood group frequencies do exist. They have lived for long periods in isolation and they are in part "marginal" geographically. Accumulation of extreme values may be striking, as in Lapps and the Ainu, perhaps residues of ancient races.

In conclusion, it becomes probable that present blood group distributions can tell us something about prehistory.

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MOURANT, A. E. 1954 The Distribution of the Human Blood Groups. Blackwell Scientific Publications, Oxford.

RETARDATION IN GROWTH OF CHILDREN IN EUROPE AND ASIA DURING WORLD WAR II

BY STEPHEN D. MARKOWITZ
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WORLD WAR II created an experimental situation for the study of the effects of wartime conditions on the growth of children in which environmental change provided an independent variable and consequences of this change, a dependent variable.

GROWTH OF CHILDREN

For the half century previous to World War II, there had been a tendency throughout the world for weight and height to increase within each age group from newborn to adult. This is clearly seen for age groups 13 to 18, for instance, in data compiled by the Japanese Ministry of Education (in Matsuyama, '48). This data is plotted in figures 1 and 2. The curves, except for a slight acceleration after 1921, rise steadily from 1901 to 1937, the last year used in this series. From this we can asume that the environment was changing at a fairly constant rate.

Curves made from data compiled by Matsuyama ('48) on a small group of boys are included on the same graphs for the years 1940 to 1946. The curves from 1901-1937 do not fit together with those of 1940-1946. Nevertheless, the two sets have been connected with dotted lines to indicate the corresponding age groups. Note the change of slope. In prewar years the curves are rising; in the war years the curves are falling. The curves for the war years, especially for height, are not steady, probably because of the small and inconstant number of children measured from year to year.

Similar curves were compiled by Solth and Abt ('51) for the weights of newborns from 1901 to 1948 in three German cities (Marburg, Berlin and Wurzburg) and in one Swiss city (Basel). The curves for all 4 cities show the same change of slope at the start of World War II as the Japanese study except that the fall in the curve for the Swiss city is much less severe and takes place only during the first half of the war. The curves for the three German cities fall rapidly up to 1946, one year after the war. At this time they reverse direction and rise very rapidly toward their pre-war levels. In explaining this Solth and Abt discount all influences that are not connected with the environment.

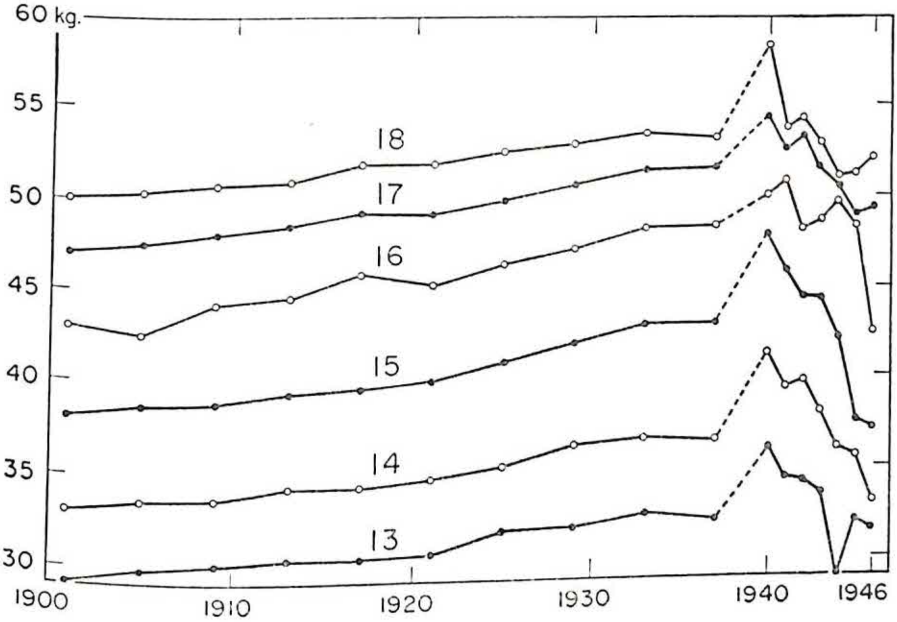

FIG. 1. WEIGHT OF JAPANESE SCHOOL BOYS FROM 1901 TO 1937; WEIGHT OF UENO MIDDLE SCHOOL BOYS, TOKYO, FROM 1940 TO 1946.

Another way of showing the effect of war environment on growth is to compare the heights and weights of children during the war with a pre-war standard. This method showed decreased height and weight in France (Douady and Trémolières, '47; Boulanger-Pilet, '46; Boulanger-Pilet *et al.*, '50; Laporte, '46; and Meiklejohn, '45), Germany (Stutte and Reinecke, '51), Greece (Valores, '46), Italy (Galeotti, '47), Belgium (Ellis, '45), China (Ni, '49), Finland (Kena-Wickstrand, '50) and Holland (Streng, cited by Hirsch, '46).

France was among the worst affected countries. In studies made in Paris, Marseilles, Nice, Lyon, Toulouse and Montpellier by several different investigators, the general outcome was a large reduction in weight and height during the war. The largest and most persistent reductions occurred among the age groups in which puberty usually begins (11-14 years of age). This retardation can be correlated with the delay of the onset of puberty that occurred generally during the war.

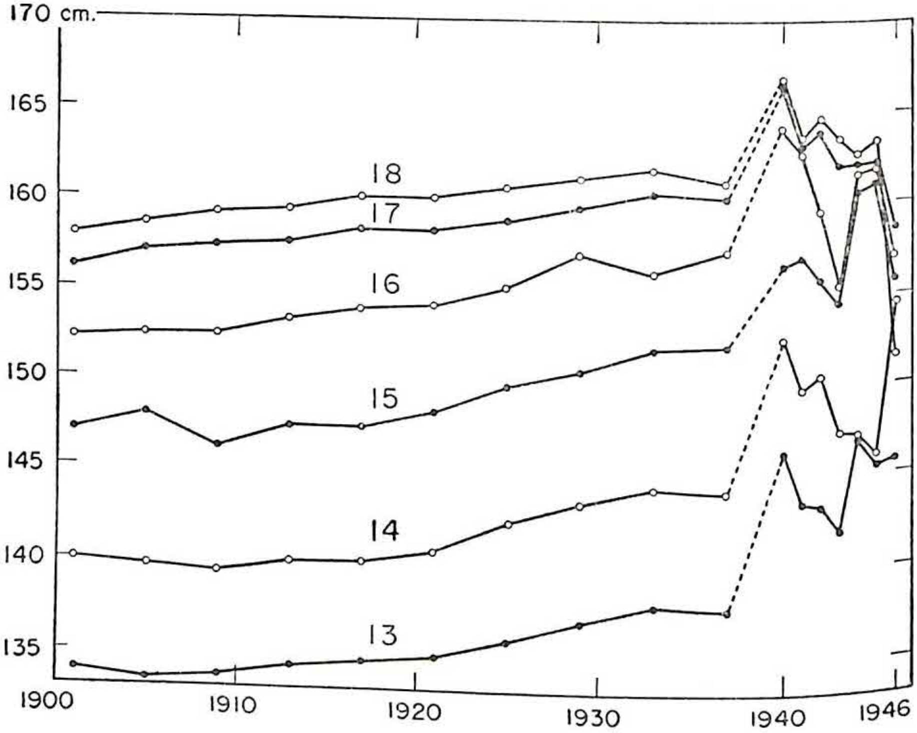

FIG. 2. HEIGHT OF JAPANESE SCHOOL BOYS FROM 1901 TO 1937; HEIGHT OF UENO MIDDLE SCHOOL BOYS, TOKYO, FROM 1940 TO 1946.

I have been informed by a French friend that food shortages in France were most acute in 1942. The food situation got progressively better in 1943, 1944 and 1945. One author, Meiklejohn ('45), states that the food situation in 1945 was up to the standards of Great Britain, while Douady and Trémolières ('47) state that nutrition, although improved, was still insufficient in 1945.

Boulanger-Pilet, Delagrange, and Trémolières ('50) compared measurements taken on the children of the *Écoles Primaires de la Seine* in the years 1938-39, 1942, 1946, and 1948. The measurements were compiled for girls and boys aged 6 to 15. The percentage changes in height and weight for the years 1942, 1946, and 1948 using 1938-39 as a standard year, are included in tables 1 and 2. The reductions in height in 1942 are very slight and take place for boys between the ages 11 and 13 (amout 1.2% reduction) and for girls between the ages of 8 and 11 (about 0.8% reduction). During the same period the boys in the 10- and the 14-year age groups had increased in height (0.8% and 0.7% respectively); the girls in the 6-year group had increased 1.8% in height. The reductions in weight were greater and not as questionable as were the reductions in height. There were reductions in weight averaging 2.9% for boys, excluding the oldest group and 3.0% for girls, including all groups. The largest reductions occurred in the older groups (in boys, age 13, 5%; in girls, age 11, 4.5%—see table 2, column 1) as in all studies where reductions in weight have taken place. There are increases in 1946 and 1948; the older groups are the slowest to recover from the deficits and girls seem to improve faster (tables 1 and 2, columns 1, 2 and 3).

I have summarized this last study in detail since it shows the least retardation in growth of any study published on French children. The reductions in height are questionable since they are small or non-existent. In respect to height, at least, this study compares with one from England (Bransby, '46) and one from Italy (Metcoff and McQueeney, '46) which did not find reductions in height and weight. Does this mean that the war environment affected only the weight of the Seine school children and not the height, and that in England and Italy there was no change in growth?

It is not necessary to find absolute reductions in height and weight to conclude that the war environment affected the growth of children. The prewar Japanese curves, or better, those for Holland (Streng, cited in Hirsch, '49) demonstrate a change in the secular trend. The average height of Dutch children tended to increase from 1938 to 1941. This increase of 6 cm was partly lost (1 to 2 cm) during the war years. Here one finds absolute reductions during the later war years if 1941 is taken as a standard of comparison but absolute increases if 1938 is taken. Thus, apparent increases in height or weight do not necessarily mean that the growth patterns were not retarded. To show that there was no change one would have to draw a continuous height and weight curve for the prewar and postwar years which did not change its slope. Furthermore, failure to change in weight probably also indicates a change in the conditions of growth if there had previously been steady year to year

TABLE 1.

Height of Children during World War II compared with Pre-war Standards (percentage differences)

COUNTRY	FRANCE														GERMANY		GREECE				ITALY		BELGIUM			CHINA		JAPAN
AUTHOR	BOULANGER-PILET, *et al.*						DOUADY AND TRÉMOLIÈRES						LAPORTE		STUTTE & REINECKE		VALORAS				GALEOTTI		ELLIS			NI		MAT-SU-YAMA
PLACE	SEINE DISTRICT						MONTPELLIER						PARIS		HESSE		ATHENS				ROME		BRUSSELS			SHANGHAI		TOKYO
STANDARD	1938–39						1938						1938		PRE WAR		1927–28				1939–41		1938–39		1938	1939		1940
COMPARISON	1942		1946		1948		1942		1944		1945		1944		1946		1942–43		1945		1945		1944		1944	1945		1945
	1		2		3		4		5		6		7		8		9*		10*		11		12		13	14*		15
	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♂	♀	♂
AGE																												
4																										−2.8	−5.8	
5																										−4.0	−3.8	
6	0	+1.8	0	0	+0.9	+0.9			−2.0	−0.2	−1.2	−0.4	−5.4	−1.6	+1.7	+4.1					−1.3	−0.5				−5.0	−6.0	
7	0	0	+0.8	+0.9	+1.7	+1.7			−1.7	−1.1	−1.5	−0.6	−1.5	−3.3	+1.8	+2.9			+3.8	+3.6	−1.4	−0.8	−3.1	−1.3		−5.1	−5.5	
8	0	−0.8	+0.8	0	+1.6	+0.8	−3.3	−4.1	−1.6	−0.5	−1.0	+0.9	−3.6	−2.9	+0.9	+2.4	+1.5	+3.0	+1.5	+3.0	−1.8	−0.3				−2.9	−3.4	
9	0	−0.8	0	0	+1.6	+0.8	−3.1	−2.3	−3.0	−0.9	−1.6	+0.4	−0.4	−2.4	+0.9	+1.9	+2.4	+1.4	+1.7	+0.3	−1.6	−0.2	−0.8	−1.2		−2.2	−2.7	
10	+0.8	−0.8	0	−0.8	+1.5	+1.5	−1.5	−1.5	−1.1	−0.2	+0.2	+0.8	−0.6	−2.4	+1.0	+1.6	+2.0	+1.9	+0.1	+0.6	−2.1	−0.2				−1.9	−3.7	
11	−1.5	−0.7	0	0	+0.7	+1.4	−3.6	−3.3	−3.4	−2.2	−3.1	−1.8	−5.5	−1.1	+0.6	+0.6	+3.6	+1.8	+0.5	−0.3	−0.3	−0.7	−2.7	−4.2		−4.5	−5.8	
12	−1.4	0	−0.7	−0.7	+0.7	+1.4	−3.5	−4.9	−3.1	−0.5	−2.9	−3.4	−2.6	−2.6	−0.1	−0.3	+3.0	+2.0	−0.5	−0.7								
13	−0.7	0	−0.7	0	+1.4	+1.4	−2.7	−2.7	−1.7	−2.7	−2.3	−3.4	−3.5	−0.9	−0.5	−1.1	+2.4	+2.7	−1.8	−1.7			−2.2	−0.7				−0.4
14	+0.7	0	+1.3	+1.3	+2.7	+2.7	−6.0	−0.8		−1.0	−5.8	−1.5	−2.6	−1.8	−1.1	−0.1	+1.8	+2.6	−3.6	−2.7					−2.6			−4.2
15													−1.3	−1.6			+1.3	+1.0							−1.0			−2.9
16													−0.9	+1.3			+0.7	+1.9							−0.2			−1.4
17																	+1.1	+2.9							−0.8			−2.6
18																	+2.0	+1.9							+0.2			−2.1

* Age to nearest birthday.

TABLE 2.

Weight of Children during World War II compared with Pre-war Standards (percentage differences)

COUNTRY	FRANCE														GERMANY		GREECE				ITALY		BELGIUM			CHINA		JAPAN
AUTHOR	BOULANGER-PILET, *et al.*						DOUADY AND TRÉMOLIÈRES						LAPORTE		STUTTE & REINECKE		VALORAS				GALEOTTI		ELLIS			NI		MATSUYAMA
PLACE	SEINE DISTRICT						MONTPELLIER						PARIS		HESSE		ATHENS				ROME		BRUSSELS			SHANGHAI		TOKYO
STANDARD	1938–39						1938						1938		PRE WAR		1927–28				1939–41		1938–39		1938	1939		1940
COMPARISON	1942		1946		1948		1942		1944		1945		1944		1946		1942–43		1945		1945		1944		1944	1945		1945
	1		2		3		4		5		6		7		8		9*		10*		11		12		13	14*		15
	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♂	♀	♂
AGE																												
4																										−2.0	−3.9	
5																										−3.9	−7.0	
6	−1.4	−1.8	+0.7	+1.0	+1.3	+1.0			−7.2	−7.4	−2.9		−15.2	−4.4	+2.9	+5.1					−0.5	−2.9				−5.1	−8.5	
7	−2.1	−2.8	+1.0	+2.6	+1.7	+2.2			−9.7	−5.0	−4.8	+5.0	−1.2	−3.0	−0.9	+3.2			−5.9	−8.6	−0.5	−4.4	−8.3	−8.8		−3.4	−7.6	
8	−2.4	−2.3	+1.4	+1.4	+0.9	+2.5	−4.0	−16.5	−2.0	−8.3	+0.4	+1.2	−7.3	−6.0	−1.2	−0.4	−12.2	−13.3	−12.5	−13.1	−3.5	−2.0				−4.6	−5.7	
9	−1.1	−3.7	+0.1	+0.4	+1.3	+1.2	−7.3	−7.4	−7.3	−5.9	−1.1	0	−5.1	−4.8	−1.4	−1.1	−9.0	−9.4	−10.9	−12.4	−3.5	−1.9	−1.8	−4.5		−4.5	−3.7	
10	−2.6	−4.5	+0.4	−0.8	+1.6	+1.7	−10.0	−6.7	−10.1	−5.4	−3.4	+0.3	−5.0	−10.2	−2.0	−4.5	−3.0	−12.5	−9.5	−15.9	−4.6	−1.3				−2.6	−3.0	
11	−4.3	−4.7	−0.1	+0.7	+0.4	+1.5	0	−13.6	−7.7	−7.6	−3.1	−5.7	−4.8	−4.0	−2.4	−5.0	−0.6	−14.9	−11.2	−20.1	−3.1	−2.8	−6.1	−13.6		−7.6	−14.3	
12	−4.5	−3.0	−1.8	−1.1	−0.3	+2.6	−16.6	−16.3	−15.2	−10.0	−12.5	−8.7	−9.5	−8.7	−4.5	−8.0	−4.2	−9.8	−14.0	−17.2								
13	−5.0	−3.7	−4.0	+1.2	−0.3	+3.7	−14.8	−12.2	−11.1	−10.2	−10.1	−7.3	−4.8	−3.3	−5.2	−7.8	−9.5	−8.9	−20.8	−21.3			−5.4	−1.6				−11.1
14	+0.8	−0.7	+4.6	+6.1	+7.5	+7.9	−12.0	−7.0	−16.8	−8.4	−10.2	−5.3	−11.2	−2.2	−7.8	−7.9	−11.6	−9.6	−25.0	−27.6					−10.7			−13.8
15													−6.8	−2.3			−5.4	−2.3							−7.9			−21.8
16													−7.1	+3.6			−0.3	+2.8							−4.8			−3.2
17																	+0.2	+3.2							0			−9.8
18																		+0.3							−0.2			−12.2
19																	+5.4											

* Age to nearest birthday.

increases. Decreases in height and weight can be taken as more positive evidence for changes in growth since the heights and weights of pre-war times certainly were not decreasing. Finally, one can easily see that the best years to use as standards of comparison are those closest to the war years. Decreases in height in France might have appeared greater if our standard of comparison had been taken around 1940 instead of 1938-39, since the former year might have had maximum values.

Two reports, one by Laporte ('46) and the other by Boulanger-Pilet ('46) deal with reductions in height and weight of Paris school children in 1944. Both seem to deal with the same group of children, but Laporte deals with ages 6 to 16 and Boulanger-Pilet, with ages 6 to 17. The standard of comparison is 1938. The percentage differences calculated from data presented in Laporte's article is included in tables 1 and 2, column 7. The maximum percentage differences in height occur for boys at the age of 11 (5.5%) and for girls at the age of 7 (3.3%) (table 1). The maximum percentage differences in weight occur for the boys at the fourteenth year and for girls at the tenth year (10.2%) (table 2); both sexes have large differences in the twelfth year. Boulanger-Pilet notes also a large difference in weight of boys at the seventeenth year (4 kg, about 8%). Laporte ('46, p. 247) explains the increase in the weight and height of 16-year-old girls by stating that "most of the older girls worked on farms during their vacations and thus obtained the benefit of better food." Boulanger-Pilet gave a similar explanation.

Douady and Trémolières ('47) in a study of children aged 8 to 14 from Paris, Marseilles, Strasbourg, Toulouse and Montpellier in 1945 found an average deficiency of 7 cm (about 4.7%) and 5 kg (about 11.6%) for the girls of 14 years, and 4 cm (about 2.7%) and 6 kg (about 14.4%) for the boys of the same age. In the 13th *arrondissement* of Paris, where the standard of life is low and where there were large deficiencies in weight and height in 1944, the reductions of 1944 had been halved in both height and weight for all age groups by 1945. In the 18th *arrondissement* of Paris, where the reductions in 1944 were nearly half those of the 13th *arrondissement*, the reductions in height are halved in 1945 but the reductions in weight remain unchanged. Douady and Trémolières relate these improvements and the improvements to be seen in Montpellier (tables 1 and 2, columns 4, 5 and 6) to the improvement in nutrition in 1945. Douady and Trémolières did not present any tables, only graphs. Since the graph for the city of Montpellier showed the differences in growth over three years, however, I felt it worthwhile to try to interpret the percentage changes from them. The graphs showed the differences in 1942, 1944, and 1945, taking 1938 as a base line. In estimating the changes I used the figures for Seine school children for the year 1938-39 as a base.

Meiklejohn ('45) in studies in Marseilles, Lyon, Nice, and Paris concludes that French children over 7 years of age show evidence of growth retardation. Stuart (cited by Ni, '47, p. 377) "concludes that probably half of the [French] children of school age show a retardation in growth."

Stutte and Reinecke give the height and weights of Hessian school children for the years 1946 to 1949. They include the data of Pfaundler which were taken as the pre-war German standards. Actually, Pfaundler's data, taken from the general German population, cannot properly be compared with data limited to Hesse. But the percentages for 1946 have been included in tables 1 and 2 to indicate the differences. The heights of the younger Hessian children increased in 1946 over those of pre-war German youth. In interpreting this, one should recall: (1) The conclusion of the French studies where the positive increments did not disprove a change in growth pattern; (2) younger children are the quickest to recover from the growth deficiencies; (3) the Hessian measurements were made one year after the war when environmental improvements would have occurred; and (4) there is no year given for Pfaundler's data. The Hessian weights are generally reduced except for the very youngest age groups (6 and 7). Note the trend in the weights as one goes from the youngest to the oldest age groups. The deficiencies increase as one approaches the years at which puberty usually occurs. This trend is also seen in the heights where the increases reach a maximum, decrease, and finally become negative.

Stutte and Reinecke also give a summary of data for the rest of Germany. They cite Mayer who found that the growth of 12-15 year old Hamburg children was severely hampered during the war. On the whole, Hamburg children were more severely retarded in growth than Hessian children, the retardation starting at the 6-year age group. They also mention that in 1946 Roninger found in Schleswig-Holstein a deficit of 5–7 cm in urban and rural children and that, in Solingen in 1945, Adam found that school children were 1–2 kg and 2–3 cm below the averages of pre-war children.

Some of the data for Greece (Valoras, '46), have been put in tables

1 and 2, columns 9 and 10, as percentages. These data do not give an accurate picture of the change in height growth during the war because standard values based on measurements made in 1927-28 may differ considerably from what would have been found immediately before the war. If one used the standard for the Seine school children, one would get all negative percentages for the age groups between 8 and 13. The weight values give a truer picture of the change in growth that followed "the country-wide famine of 1941-42. . . Measurements from the 1945 survey indicate that the continued undernutrition had affected height growth as well as weight and the children were shorter and lighter than those of similar age measured two to two and one-half years previously" (Valoras, '46, pp. 215 and 228). Thus by 1945 height was definitely affected and Valoras points out that it was probably affected in 1942-43 by considering "the consistent decline in height increase for boys from age 11 to 17 years, normally a period of rapid growth" (Valoras, '46, p. 228).

One study in Italy failed to find changes in growth (Metcoff and McQueeney, '46). We have already concluded, however, that a constant pattern probably represents a change from a pattern which is graphed as a rising slope. For another Italian study which reveals growth reductions see tables 1 and 2, column 11. In addition to the reductions in weight and height, thoracic circumference was reduced 3.2 and 4.4% in males and females respectively between the ages of 6 and 11. Galeotti ('47, p. 295) concludes: "Such deficiencies should be for the average of children in Rome really greater than appears from the above figures inasmuch as the children studied in 1939-41 belonged to the poorest social classes. The growing conditions of such children are in fact normally worse than those relating to a great number of children chosen at random (as it is in the 1945 survey)." This would imply that the wealthier social classes had better environmental conditions for growth during as well as before the war. In the case of nutrition, this was true in certain other countries as well. A Hong Kong girl relates that the rich were always able to get food during the worst shortages of the Japanese occupation. Ellis ('45) and Meiklejohn ('45) tell about the black markets that operated in Belgium and France, respectively, during the war and for a period after. However, Boulanger-Pilet ('46) notes that some of the poorest Parisian families lived on the outskirts of the city and had the advantage of bicycling to the nearby country for food.

Ellis ('45) found reductions in height and weight of the children of the working-class community of Andelecht in Brussels. The comparison year is 1944 and the standard is a random sampling of the community for 1938-39. Since the percentages are based on data that is subject to sampling errors they only "can be taken as indicating . . . an average diminuation of both height and weight in 1944 as compared with 1938-39 . . ." (Ellis, '45, p. 103). Ellis also compared the heights and weights in Andelecht for the years 1942, 1943, and 1944. He found that the "working-class children living at home have shown a gradual improvement in weight from 1942 to 1944, and that their height (with the possible exception of those reaching the normal age of puberty during the war) has been little affected." 1942 was the year of serious food shortages. By 1944, these shortages were corrected to some extent "by distribution of herrings, by an increased supply of potatoes, and finally by organization of the black market." (Quoted by Ellis from Bastenie and others).

Ellis follows the growth of 10 girls from the age of 7½ in 1939 to 12½ in 1944. The height and weight curves are graphed along with the standard of 1934 (compiled in the "period of severe economic depression in 1928" but published in 1934) for the corresponding ages. The height and weight curves follow the 1934 standard until 1942 (girls at the age of 10½) and then fall below the standard in 1943 and 1944, the last year computed, when the girls were aged 12½. Ellis indicates severe deficiencies in height and weight of employed telegraph boys (column 13) in 1944 as compared to the 1938-39 standard for employed boys. These deficiencies take place between 14 (the earliest year computed) and 16 and they amount to 2.6% in height and 10.7% in weight for the 14-year olds. Finally, he concludes that "adolescents . . . suffered more than the younger age group and a study of 106 adolescent girls indicates a retardation of puberty in a significant proportion of those living at home. . . ." Further, he shows by the device of three superimposed weight curves that there was a "marked delay in 1942 (following the severest period of rationing), a moderate delay in 1943 and an approximately normal onset of puberty in 1944."

Ni ('49, p. 680) found reductions in the heights and weights of "over 1,000 Shanghai school children 4 to 11 years of age, in 1945 . . . [as compared with] those of school children studied in 1939 for the corresponding age groups . . ." (tables 1 and 2, column 14). In this case it seems that the children aged 5, 6, and 7 and those aged 11 were the worst affected. Ni ('47, p. 378) notes that the 6- and 7-year olds are "more interested in games and muscular activities and, therefore, require more food than in younger years [while at the same time] . . . their actual intake of food is more dictated by the taste, flavour and appearance of a meal, by the emotional state of the children, and by the patience, experience, and tact of the parents than either by the body requirements or by the parents' instruction. . . ." This may be the reason why the younger groups show retardations of the same magnitude as the oldest group. In European countries, although the rationing practices favored the younger groups, large deficiencies can sometimes be found in the 6- and 7-year olds. For instance, see column 7 of tables 1 and 2.

In column 15 of tables 1 and 2 the percent differences for Japanese boys (Matsuyama, '48) are represented. These differences are based upon the data from which figures 1 and 2 were made except that only the years 1940 (the standard year) and 1945 (the comparison year) are used. This column forms a basis of comparison between the continuous curve and percent of absolute difference methods. Matsuyama also gives data for thoracic circumference for the entire 13- to 18-year old male Japanese population from 1901 to 1937 and for a small group of 13- to 18-year old Tokyo boys from 1940 to 1946. The percentage decrease in 1945 as compared to the standard year 1940 averaged 3.2% with a maximum decrease in the 13-year olds (8.3%).

BIRTH WEIGHT

In table 3 we have the percentage reductions in birth weights for Holland, Russia, Germany, and a neutral country, Switzerland. The percentages listed under Solth and Abt were calculated from estimates derived from the curves which they published.

Leningrad, Russia, seems to have been one of the most severely affected, having maximum differences of 18.3% and 16.4% for males and females, respectively, in the period January-June 1942, one year from the standard. These decreases were noted during the siege of Leningrad (1941-1942) by Antonov ('47, p. 254):

> The fall in the average birth weight was much greater in the first half of 1942, that is among mothers who suffered from hunger during all or nearly all of their periods of pregnancy. . . . Almost equal decrease in average birth weight of children carried to term was observed in other maternity clinics in Leningrad. In the Sneginev clinic, the average decrease was 500 gm (about 15%). In the obstetric-gynecologic clinic of the Second Leningrad Medical Institute the average weight in 1942 was 410 gm less than in 1940.

Dean ('50) found in Wuppertal, Germany a reduction 185 gm (about 5.4%); the shortening of the gestation period could account for 45 gm (about 1.3%). This reduction in birth weight took place "in 1945 when the food situation was at its worst."

Smith ('47, p. 236) finds 8-9% reduction in the weight of Rotterdam newborn in 1945 compared to "the weight in normal times, the percentage being only slightly greater in the heavier infants . . . than in the light ones. . . ." Normal times presumably means the pre-war winter 1938-39 in the same hospital in Rotterdam. In table 3 I use a war period immediately preceding the period of "nutritional stringency," and therefore the percent difference shown is a smaller value (7.1%). Schaible ('49) noted a 7.3% reduction in newborn of Reutlingen, Germany for 1945-1947 as compared to 1938-1942. Belgium and Italy seem to be exceptions as far as absolute differences in birth weight are concerned (Ellis, '45; Pozzan, '51).

The general conclusion of all the investigators was that retardation in growth occurred during the war, but none of the investigators were successful in defining the specific factors which controlled the change in growth. The most they did was to describe the events of war and state that these events affected the development of the child. In attempting to classify environment, one is only concerned with that part of the environment that affects growth. We must link the specific parts of the environment with the body reactions.

To do this, one could start with the body reactions themselves. All body reactions can be equated with the total energy of the organism. This total energy encompasses the energy of growth and the energy needed to keep alive. The energy of growth is of secondary importance to the energy needed to keep alive since an organism must be alive in order to grow. Therefore, in times when the energy produced by the body is lower than normal, the energy of growth would be expected to suffer for the sake of conservation of adequate energy to keep alive.

If the war data is reviewed, it will be seen that events were correlated with reductions in weight and height. One of these, the increase in the age at which puberty begins, has been noticed by several investigators (Ellis, '45, Douady and Trémolières, '47). The energy needed to cause the physiological changes that occur with the onset of puberty is energy of growth. During the war the lack of food and the inefficient use of existing food, due to psychological conditions, would produce a limited amount of body energy. What energy was produced by the body would

TABLE 3.

Birth Weight of Infants Born during World War II compared with Pre-war Standards (percentage differences)

COUNTRY AUTHOR CITY STANDARD COMPARISON	Russia Antonov Leningrad January—June, 1941						Holland Smith Rotterdam Oct. 1—Nov. 15, '44 Nov. 16, '44— Feb. 14, '45	Dean Wuppertal 1937–38 1945	Germany Solth and Abt			Schaible Reutlingen 1938–42 1945–47	Witzerland Solth & Abt Basel 1939 1945
	Jul.—Dec. '41		Jan.—Jun. '42		Jul.—Dec. '42				Berlin 1939 1945	Marburg 1939 1945	Wurzburg 1939 1945		
	♂	♀	♂	♀	♂	♀							
	−3.0	−2.4	−18.3	−16.4	−7.1	−12.5	−7.1	−5.4	−2.1	−3.2	−2.2	−7.3	−0.8

be used in keeping the body alive, not in bringing about the onset of puberty. In many countries, notably Greece, Holland, and Russia, 50-70% amenorrhea was prevalent during the worst food shortages (Antonov, '47; Smith, '47; Valoras, '46). As in the case of pubescent growth, the decline in menstruation cycles would conserve body energy in the female.

If we begin to understand the processes of reactions within the body, we can proceed to look for the specific stimuli of these energy producing reactions. As in the case of the body reactions, we can divide the environmental stimuli into primary and secondary. Under primary we would include such things as nutrition and respiration since these are necessary for life. Hirsch ('49) in his study of Jerusalem children during the Arab-Israeli War also mentions the amount of sunlight as a factor. Psychological phenomena would be secondary.

We now begin to see the inadequacy of the experimental situation created by World War II. We have correlated the reduction in growth with a reduction in body energy output. However, it is impossible with the present data to separate the effects of nutritional and psychological stimuli on wartime growth. The relative importance of the two stimuli is unknown. Most of the workers considered the nutritional stimulus. Nutrition may be the most important factor, but to consider nutrition alone is to disregard the individual as a dynamic organism responding to stimuli other than fuel. Psychological factors may also have been involved. Conditions of war create stress and this may affect appetite, efficiency of digestion and absorption, and efficiency in utilization of absorbed food. Banning (cited by Keys *et al.*, '50, p. 92) ascribed the weight loss in the population of Holland in the first months of World War II to psychological factors "since the food supply was not greatly deficient."

The heights and weights of English school children generally remained constant during the first years of World War II. If the English heights and weights were increasing in pre-war times, this constancy would indicate a change in growth patterns. And, if this be true, this change cannot be correlated with nutrition since England suffered no more than a "slight underfeeding" (Keys *et al.*, '50, p. 17). From 1943-1945, heights and weights of English school children were above pre-war levels (Bransby, '46). This may indicate an adjustment to the psychological stress.

SUMMARY

World War II has provided us with an experimental situation for the study of growth. Although few studies permit one to draw a continuous curve of growth throughout this period for a homogeneous group of children over a span of time, several permit one to plot two or three points and to consider absolute differences. From studies of heights and weights of children and weights of newborn infants in Japan, China, England, France, Germany, Switzerland, Italy, Greece, Belgium, Holland, Finland and Russia one reaches the general conclusion of a retardation in growth during World War II. The role of nutritional (part of the primary stimulus) and psychological (part of the secondary stimulus) factors cannot be separated, however. Although it is clear that nutrition affects growth, it is not possible to isolate the factor of food shortage in the war situation. When and where food was scarcest, growth was usually most retarded, but there, also, conditions for emotional upset were at a maximum. The importance of psychological factors is still to be determined.

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FINGERNAIL GROWTH IN THE HUMAN SUBJECT

RATES AND VARIATIONS IN 300 INDIVIDUALS

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GROWTH, other than neoplastic, is probably one of the more acceptable attributes of health. But, except for the physiological phenomena of pregnancy and the reparative responses to stress, growth—as distinct from development—is not a measurable characteristic of most "normal" adult human tissues. The maintenance and replacement of epithelial surfaces, however, is at least a related activity, and the skin provides an accessible area for observing this process. Its variable appendages, the hair and nails, have been intensively studied by anthropologist and clinician alike, as evidenced by numerous published descriptions of their characteristics in health and disease.

These observations have been almost exclusively qualitative—particularly as concerns the fingernails. Moreover, most of the occasional, recorded quantitative nail growth studies have related to only a few or even single patients with local or systematic disorders. Sharpey-Schafer ('30), Wigand ('37), Sunderland and Ray ('52), and, in this country, Edwards ('38) are among the principal recent contributors to this limited available information. Beans' ('53) 10 year self-study constitutes a uniquely long series of observations, while the largest collections of data on the subject appear to have been assembled by Clark and Buxton ('38) and Gilchrist and Buxton ('39). Although these last involved a relatively narrow age range (10 to 23) over evidently short periods of time, they seem the most authoritative basis for current concepts of nail growth rates in the normal human subject.

The present inquiry was designed to provide further information concerning the growth rate of fingernails, particulary to document intra- and inter-individual variations and to determine the possible influence of such factors as age, sex, intercurrent illness and environmental change. A second objective was related to the concept that significant departures from established growth patterns might reflect pathological states and that the practical demonstration of these irregularities might facilitate the early detection or the rate of progression of disease.

MATERIALS AND METHODS

Fingernail growth was measured in 300 individuals, including 148 medical students and 63 student nurses. The remaining subjects were predominantly professional, technical and secretarial personnel, supplemented by two family groups. In all, 293 subjects were considered ostensibly "normal." Of the others, two had diabetes mellitus, two rheumatoid arthritis (one on intermittent steroid therapy), two peptic ulcer syndrome (one post-gastrectomy), and one an active, chronic suppurative osteomyelitis. Relevant personal data were provided by questionnaires, supplemented in most instance by health records.

There were in the study 179 males and 121 females, including one set each of identical and fraternal twins. The age range was 4 to 67 with a median of 21 and a mean of 25.6 years. The average period of observation was 154 days, the median being 176 and the range 25 to 457 days. Ten individuals were observed for a period of at least one year. There was a total of 991 separate observation periods, ranging from two to 8 weeks, with an average interval of 46 days, providing a cumulative total of approximately 46,200 person-observation days. Since from two to 10 nails were measured for each subject, the number of nail-observation days was obviously greatly in excess of this figure.

Growth rate was determined by measuring the distal progress of a transverse mark, inscribed with an "ampoule" file at the convex margin of the lunula. A fine-pointed, "E.C.G." type calipers was employed to span the intervening space. The distance was then measured off on a blocked steel rule, and the result converted into a metric reading. All measurements were made by one of two observers, with readings being satisfactorily reproducible.

RESULTS

Results are summarized in tables 1 and 2 and in figures 1-3. All growth rates are recorded in microns per day, and represent measurements on first digits only, these being common to all subjects in the study group.

TABLE 1

Nail growth rates for various categories of subjects [1]

	MALE					FEMALE				
	Number	Mean	Standard Deviation	Coefficient of Variation	Range	Number	Mean	Standard Deviation	Coefficient of Variation	Range
Total [2]	179	117.0	16.6	14.4	74–168	121	111.9	17.2	15.4	74–155
Under 10	2	112.0	—	—	107–117	0				
10–29	149	119.6	15.3	12.8	84–168	80	116.6	16.7	14.3	79–155
30–39	12	114.8	9.7	8.4	99–132	17	103.2	16.6	16.1	74–145
40–49	9	101.1	7.5	7.4	88–117	12	103.3	10.5	10.2	86–122
50–69	7	88.9	9.4	10.6	74–102	5	101.2	13.2	13.0	84–120
Nail Biters	29	128.2	18.0	14.0	86–168	10	135.9	15.2	11.2	107–155
Non-Biters	113	117.4	13.7	11.7	84–155	59	115.2	14.8	12.8	91–147
Overweight [3]	15	116.3	20.3	17.5	84–168	8	124.3	11.9	9.6	109–145
Underweight [3]	16	125.5	16.7	13.3	91–155	15	117.0	17.1	14.6	94–152
Heavy [4]	15	121.3	13.2	10.9	94–137					
Light [5]	14	125.8	15.4	12.2	107–155					
Tall [6]	21	122.7	17.3	14.1	91–163					
Short [7]	21	119.0	18.9	15.9	84–168					
Weight Gaining						31	119.4	15.9	13.3	94–152
Weight Losing						13	122.2	20.7	16.9	94–155
Smokers [8]	25	111.1	14.6	13.1	84–155	11	113.3	10.5	9.3	102–130
Non-Smokers	71	120.7	14.1	11.7	89–168	36	116.4	14.9	12.8	89–145
Smokers, Non-Biters [8]	20	111.0	15.6	14.1	84–155	11	113.3	10.5	9.3	102–130
Non-Smokers, Non-Biters	55	117.3	12.1	10.3	89–145	33	114.7	14.1	12.3	89–145

[1] All figures in microns per day for first digits only. Except for the age data and totals, all data are from medical students and student nurses only and from within the age range 10–29.

[2] Includes three females whose specific ages were not provided.

[3] Outside the limit indicated as normal for height and sex in tables of the Metropolitan Life Insurace Company.

[4] Over 190 lbs.

[5] Under 150 lbs.

[6] Over 72 inches.

[7] Under 68 inches.

[8] Includes only those consuming 10 or more cigarettes per day.

The overall mean growth rate was 115 ± 17 microns per day, with a range of 74 to 168. The rate was relatively regular in most individuals, the average deviation (of single readings from each individuals's respective mean) being 5.5 ± 6.5 microns. There was considerable variation between the hands, but no apparent relationship to so-called "handedness." Of 209 subjects in whom this comparison could be made satisfactorily, the nails on the right hand grew more rapidly in 37 (17.7%), and those on the left in 33 (15.8%). The hand rates were equal in 59 (28.2%) and variable (in different digits and/or at different times) in 80 instances (38.2%).

TABLE 2

Mean intra-individual variation in nail growth rate for various age and categorical groups[1]

Group	Sex	Number[2]	Mean	Standard Deviation	Coefficient Variation	Range[3]
Total	M&F	243–824	5.5	6.5	118.2	0–42
Total	M	149–439	4.3	4.5	104.7	0–19
Total	F	94–385	6.8	8.1	119.1	0–42
Under 10	M	2–10	10.2	6.9	67.6	3–12
10–29	M	123–329	3.6	3.7	102.8	0–16
30–39	M	11–56	7.3	6.5	89.0	2–14
40–49	M	7–26	4.3	3.8	88.4	0–6
50–69	M	6–18	4.9	4.0	81.6	2–8
10–29	F	66–281	6.4	8.2	128.1	0–42
30–39	F	13–50	5.6	5.7	101.8	0–10
40–49	F	9–24	9.8	10.1	103.1	2–37
50–69	F	6–30	9.8	6.7	68.4	3–13
Nail biters	M	20–54	4.5	3.4	75.7	10–93
Nail biters	F	8–32	6.2	7.4	119.4	25–80
Non-biters	M	99–261	3.5	3.5	100.0	0–16
Non-biters	F	50–209	6.1	8.4	137.7	0–42
Weight-gaining	F	22–109	7.3	10.5	69.4	3–42
Weight-losing	F	10–41	6.7	7.6	122.6	2–17
Overweight	M&F	20–65	4.4	5.1	115.8	0–10
Underweight	M&F	22–85	6.9	11.1	161.2	1–42
Smokers	M&F	30–88	4.3	4.2	97.7	1–9
Non-smokers	M&F	88–284	4.6	4.2	91.3	1–16

[1] All figures in microns per day, for first digits only.

[2] Includes all subjects with two or more readings. First number indicates total subjects; second indicates total readings.

[3] Indicates mean intra-individual variations for given subjects.

Of 136 subjects in whom the relative rates of nail growth could be satisfactorily determined, the rate was most rapid in the third digit in

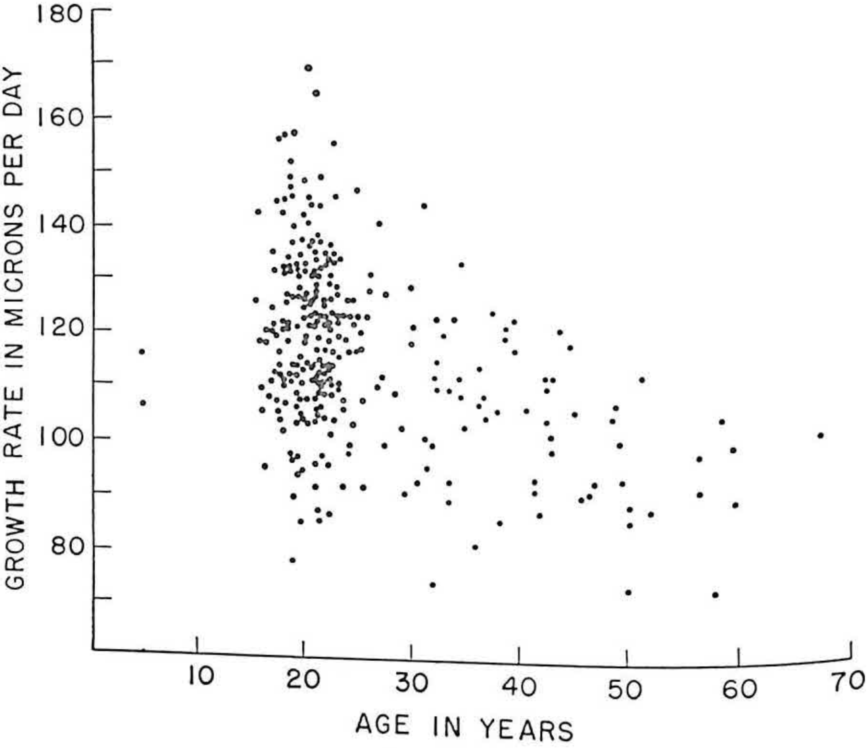

FIGURE 1.

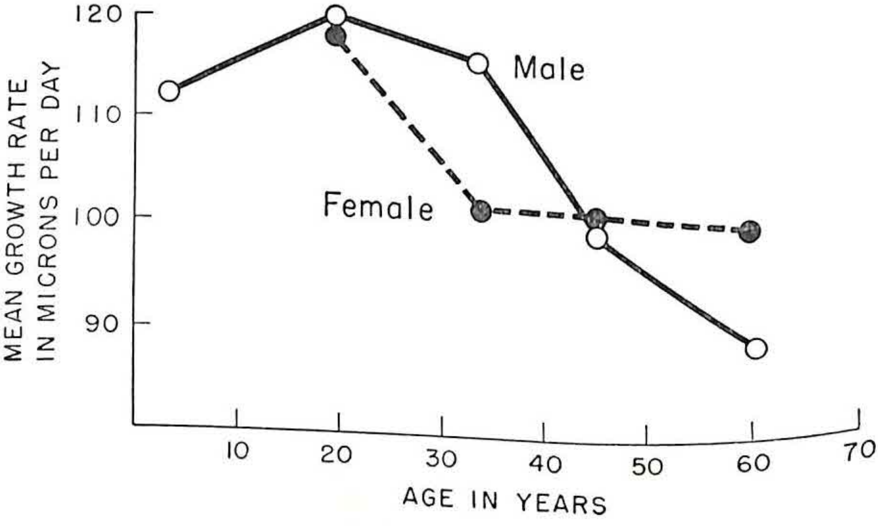

FIGURE 2.

91 (67%) and in the first in 16 instances (11.8%). The rates were identical in the first and third digits in 17 individuals (12.5%) and variable in 6 (4.4%). In 6 other persons growth was most rapid in one of the other digits.

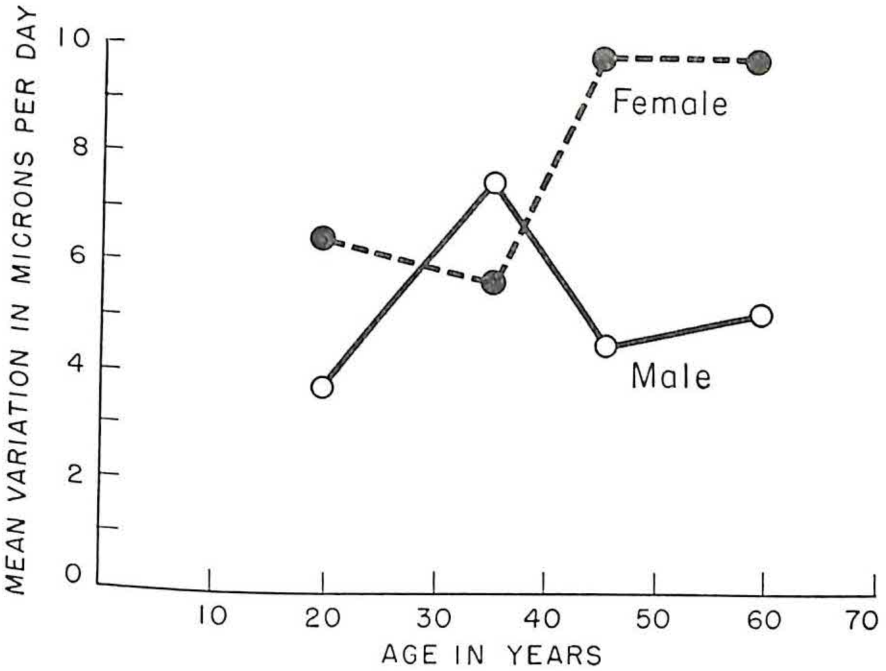

FIGURE 3.

In addition, the following general impressions were formed:

There was no seasonal variation in nail growth. The rate was unaffected by minor intercurrent illness, change in work or dietary habits, or by moderate stress situations (*e.g.*, scholastic examination periods). Subtotal thyroidectomy for adenoma in one individual did not appreciably modify growth, and the rates among the 7 patients did not differ significantly from those of ostensibly normal persons in the same age groups. Rates were similar in the identical and fraternal twins, although an older sibling (same sex) of the latter pair showed much more rapid nail growth.

More specifically, there was no statistically significant difference in average nail growth rate between tall and short, heavy and light or overweight and underweight individuals; nor between those gaining and those losing weight during the period of observation. Overweight subjects exhibited less intra-individual variation than those below normal weight, as did those losing compared with those gaining weight, although these differences were not statistically significant.

The growth rate was significantly greater (P less than .001) among nail biters than among non-biters—so that other comparisons involved correcting for this factor.[1] For example, the crude growth rate among non-smokers was significantly greater (P less than .01) than among smokers, but the difference was not statistically significant after the biters had been eliminated from each group (possibly an effect of sample size in part, since each group included nearly the same proportion of individuals admitting this habit).

There was no apparent relationship of overall nail growth rate or variability to the subject's ordinate birth status, nor to his mother's age at the time of his birth. Likewise, there was no apparent relationship between these characteristics and individual life expectancy (as estimated from forebear experience), nor between them and a family history of cancer, hypertensive and coronary artery disease,[2] or diabetes mellitus.

There was no apparent difference between the nail growth rates of white and non-white subjects, but the latter group was too small for valid conclusions. The rate did, however, seem to relate to the factors of sex and age, being generally more rapid in males and in younger individuals. These variations are depicted in figure 2.

The mean growth rate for all males was 117 ± 17, with a range of 74 to 168 microns per day. For females it was 112 ± 17, with a range of 74 to 155. This difference was statistically significant (P less than .01). In the age group 10 to 30, representing a majority of all subjects, the average rates were 120 ± 17 and 115 ± 17 for males and females, respectively. This difference was not statistically significant. However, a significant fall (P less than .01) in the average female rate (to 103) in the group 30 to 40 years of age, compared to only a slight contemporaneous decrease in the males, resulted in a significant difference (P less than .05) between the sexes in this decade. Successive, statistically significant drops in the male rates for the groups 40 to 50, and 50 to 70 years of age produced a mean of 89 among these last grouped subjects. This was substantially below the average of 101 for their female counterparts, although the gap was not statistically significant. The differences between mean growth rates for the groups under 40 and those over 40 were statistically significant (P less than .001) for each sex individually, and for both sexes considered together.

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[1] The incidence of nail biting among overweights (34.8%) was significantly greater (P is less than .05) than among underweights (9.7%); it was also significantly greater (P less than .05) among those losing weight (46.2%) than among those gaining weight (16.1%) during the period of observation. Individuals with family histories of coronary artery disease likewise included a relatively high incidence of nail biters.

Other mechanical stress, *i. e.*, the regular application of nail polish and remover seemed not to effect nail growth rates.

[2] The mean intra-individual variation was not significantly increased among subjects with a family history of hypertension, but the ratio of individual mean variation to individual overall mean growth rate seemed highest in this group.

Inter-individual variation, as reflected in the coefficients of variation, was greater for females than males in all age groups.

Intra-individual variation, as represented by average variations from respective individual means, was likewise greater among females than among males (figure 3), but appeared to have no overall relationship to age. The mean variation for all males with two or more individual readings (4.3 ± 4.5) was significantly different (P less than .002) from that for all females (6.8 ± 8.1). It was also significantly lower in each age group, except in persons 30 to 40, among whom, unaccountably, the females exhibited less intra-individual variation. Again, the females generally showed more inter-individual differences, with greater coefficients of variation in all but the 50 and over age group.

COMMENT

In general these results are in accord with others previously reported, notably in respect to the average growth rate of about 0.1 millimeter per day, the lack of relationship to so-called handedness and the apparent propensity for more rapid growth of the nail of the third digit in most instances. The present observations, however, appear not to support the impression of seasonal growth changes, emphasized by Clark and Buxton ('38) and Gilchrist and Buxton ('39), but are in agreement, rather, with Bean's ('53) similar failure to demonstrate such a relationship during his 10 year self-study. They likewise seem consistent with his impression that the nail growth rate decreases with age and is essentially unaffected by changes in work habits, physical environment or by mild, intercurrent infection—although it does undeniably vary at times for reasons not yet identified. The considerable inter-individual variation evidenced in the present group is in accord with the large-scale observations of Clark, Buxton and Gilchrist, whose impressions of a lack of consistent relationship between nail growth rate and age or sex may have been due to the relatively narrow span of early years represented by their two series. Berthold's contentions, as noted by Edwards ('38) and based on a wider age range of subjects, would seem to support further the concept of some relationship of nail growth to the period of life.

Intra-individual variation, in contradistinction to differences between persons, apparently has not been emphasized previously in the literature. In this study the wide variety of personal growth patterns observed among the subjects and the apparently real difference between the sexes noted in respect to constancy of rate, enhance the impression that these individual irregularities may ultimately prove of more biological significance than average nail growth rates.

The implications of these observations are not readily apparent—apart from a possible endocrine cyclic component operating in females (but evidently not limited to the premenopausal group). At present, individual nail growth rates seem not to be valid indices of personal health or to comprise a likely, practical means of detecting early deviations from the normal or satisfactory state of general wellbeing. However, the notable inter- and intra-individual variation may actually reflect fundamental metabolic differences, some conceivably at the "end organ" level, among normal functioning persons. The possibility that nail growth patterns may be related to other, more basic, biological phenomena governing the overall physical state and the future expectations of the individual warrants further investigation.

SUMMARY

Fingernail growth rates were measured in 300 individuals, including 293 ostensibly normal subjects and 7 with chronic disease.

The overall mean growth rate of 119 microns per day was consistent with impressions provided by other studies, although considerable intra- as well as inter-individual variation was observed, notably among females.

In general, nail growth seemed more rapid in the third digit, in males, in the younger age groups and in nail biters. It was questionably slower among smokers than non-smokers.

Nail growth rate seemed unrelated to such factors as season, handedness, body height and weight, minor changes in living habits and mild intercurrent illness. The 7 individuals with chronic disease showed no

significant difference in growth rate from their ostensibly healthy counterparts.

The explanation for these observed variations is not apparent, and further investigation of nail growth patterns is indicated—especially studies of intra-individual change—for possible basic biologic implications and practical clinical applications of these easily measured phenomena.

The author acknowledges the assistance of Miss Marie Leogrande who performed many of the fingernail measurements.

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THE ABSENCE OF THE SICKLE CELL GENE IN SEVERAL MELANESIAN SOCIETIES AND ITS ANTHROPOLOGIC SIGNIFICANCE

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INTRODUCTION

WITHIN the last few years numerous papers have appeared concerning the sickling phenomenon (Pauling *et al.*, '49, Neel *et al.*, '51, Neel, '51b), the inheritance of the sickle cell gene (Beet, '49, Neel, '49, '51a, Lehmann, '51), and the distribution and ethnographic significance of this blood dyscrasia (Lehmann and Raper, '49, Hiernaux, '52, Foy *et al.*, '52). Perhaps the clearest statement is one by Singer ('53), in which he summarizes the information available on the sickle cell gene, as well as raises certain theoretical problems important to racial anthropology. The sickle cell (Neel's homozygous-heterozygous theory of inheritance is accepted in this paper) is widely distributed throughout Africa (Singer, '53) and has recently been reported among three Veddoid tribes of Central India (Lehmann and Cutbush, '52). The gene also has a sporadic frequency in other parts of the world where Negro admixture may have occurred (Caminopetros, '52, Dunlop and Mozumder, '52, Powell *et al.*, '50, Allison, '53). This information, coupled with a suggestion by Dr. Neil Tappen that the author check for sickling while in Melanesia, was responsible for the survey reported here.

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* Physical anthropologist with an ethnographic expedition to New Britain jointly sponsored by the Museum and Department of Anthropology of the University of Pennsylvania. Additional funds were contributed by the American Philosophical Society and the Tri-Institutional Pacific Program. The author's participation was aided by a Wenner-Gren Foundation pre-doctoral fellowship.

The purpose of this paper is threefold: 1) to report the absence of the sickle cell gene among several Melanesian societies; 2) to consider the anthropologic implications that may be adduced with respect to the genetic affinities of the Oceanic Negro in the light of this new evidence; and 3) to discuss what significance the sickle cell gene affords the student of race in view of the most recent hypotheses about this abnormal blood characteristic.

MATERIALS AND METHODS

Since some doubt has been registered about the reliability of the techniques and results of several investigations (see especially Singer, '53), the author spent two weeks before departing for Melanesia in the Department of Hematology at the Children's Hospital of Philadelphia studying the sickling phenomenon. During this period he had the opportunity to observe blood samples from individuals with the sickle cell trait and sickle cell anemia. He was also taught a simple and reliable technique for eliciting sickling in which a reducing agent, sodium metabisulfite, is employed.

The field method consisted of dissolving a three-grain tablet of sodium metabisulfite in approximately 10 cc of sterile water. Fresh preparations were made daily to ensure maximum strength of the solution. Recently, Allison ('54a) has advocated this procedure when using chemical reducing agents in the tropics. One drop of blood was obtained from each individual by finger prick and placed on a glass microscope slide. Immediately a drop of metabisulfite solution was added to the slide and mixed thoroughly. A coverslip was then placed over the preparation and ringed with a thin layer of petroleum jelly, which was melted with a match flame and then permitted to solidify. The slide was then ready for microscopic study at various intervals. All slides were checked for sickling at 15 minutes, 24 hours, 48 hours and, when feasible, at 72 hours, although in a few instances the blood had deteriorated and the erythrocytes were not discernible after 72 hours. Consequently, if no sickling had occurred by the 48-hour check the individual was considered negative for drepanocytosis (sickling).

Blood smears were also made for each individual. The cells were preserved by pouring methyl alcohol over the slide. These preparations were returned to the Children's Hospital, where Dr. Patraula Typaldou examined them for sickle cells and other blood features. This study was conducted six months after the smears were collected.

A total of 277 blood samples were collected and studied in the field.

Of these, 156 were from the West Nakanai, who live along the Hoskin's Peninsula, which is located on the north central coast of New Britain in the Territory of Papua and New Guinea. The remaining 121 slides were collected and examined at a small hospital unit in Talesea, which is a government outpost situated about 75 miles northwest of the West Nakanai area. Here we had the opportunity to investigate members of various tribes in the vicinity when they came into Talasea for medical attention. In fact, the series include three "police boys" from the Sepik River regions on New Guinea, as well as natives from Vitu and Bali islands which lie off the north coast of New Britain. We were quite particular to exclude from both series all near relatives; in other words, not more than one member of a family was included in the survey.

TABLE 1

Individuals separated as to tribe, age, and sex

TRIBE	ADULT		CHILDREN	
	MALE	FEMALE	MALE	FEMALE
West Nakanai	99	44	7	6
Bakovi	28	11	5	1
Kambi	23	15		
Bulu	8	6	2	1
East Nakanai	2	2	2	
Central Nakanai	1			
Taulai (Rabaul)	2		1	
Banale	1			
New Guinea (Sepik R.)	3			
Bali Island	1		2	1
Vitu Island	2		1	
Total	170	78	20	9

The separation of both series into tribe, sex, and age categories may be seen in table 1. The division between adults and children is somewhat arbitrary since chronologic age is not recorded. We could only estimate the individual's maturational status as registered by his or her acquisition of certain well-known secondary sexual characteristics plus Hellman's dental ages. In short, if an individual lacked any indication of axillary hair or beard and his third molar teeth were unerupted, he was classified as a child.

RESULTS AND DISCUSSION

There was no indication of sickle cell anemia or sickle cell trait in the slides examined by us in the field or in the returned smears studied by Dr. Typaldou. Unfortunately the sample is rather small, but we feel justified in stating that at least among one Melanesian society, the West Nakani, the sickle cell gene is absent.

Before considering the anthropologic implications of this finding, attention will be directed to other studies which have been conducted in this general area for the sickle cell, and which appear to corroborate our data. During a disease survey of the Malayan aborigines, Polunin ('52) did not find sickling to be present in a group of 150 Malayan Negritos. Sneath ('54) reports the absence of the sickle cell in both the Malayan Negritos and aboriginal Malays. His samples consisted of about 100 members of each group. According to Horsfall and Lehmann ('53) there was no evidence of sickling among 52 Onges of the Little Andaman Islands and 16 Andamanese of mixed Negrito ancestry studied by Lehmann. They also report the absence of the sickle cell gene in 72 Australian aborigines. Of these, 57 individuals were of unmixed Australian ancestry and presented many Carpentarian (Veddoid) characteristics. Simmons *et al.*, ('53) also failed to find any evidence of the sickle cell in 125 random adult blood samples obtained from Western Australia. These data, coupled with the results presented herein, strongly suggest that the sickle cell gene, wherever its origin or origins may have been, did not pass any farther eastward than India.

Of the many racial elements represented throughout Melanesia today the influence of the Oceanic Negrito and Negroid is the most widely disseminated. The Mongoloid and Caucasoid contributions are also present in various parts of the area but are of little consequence to the hypotheses of the present paper. These 4 components then form the matrix of the racially blended populations inhabiting New Guinea and the remainder of Melanesia. The origins of the Oceanic Negrito and Negroid are shrouded in antiquity and will probably never be completely unraveled. Most authorities would agree, however, that they are genetically related to their African counterparts, the African Pygmy and Negro. If this relationship can be proven and if the sickle cell gene is actually indicative of Negro ancestry it is incumbent upon us to elucidate the enigma. Birdsell ('49, p. 3) is most emphatic concerning the origin of the Oceanic Negrito; he writes, "If there is any safe inference in the field of racial anthropology, it is the Oceanic Negritos must be considered as very closely related genetically to the Negritos of the Congo Basin in tropical Africa." Earlier, Howells ('43, p. 43) was somewhat less absolute in his opinion, but nevertheless stated that "It is very difficult to doubt that the pygmy peoples of New Guinea are fundamentally of the general Negrito stock." In the second edition of *Introduction to Physical Anthropology* Montagu ('51, p. 315) concurs rather ambivalently with this idea, for he writes, "The superficial resemblances of the pygmies have inclined many students to consider them as closely related. Their peculiar distribution would tend to support this view." The consensus, then, is that these two groups are genetically related, but differences of opinion arise with respect to the degree of genetic affinity between them.

When the Oceanic Negro is considered, his relationship to the African Negro is more ambiguous. Here there is a definite dichotomy. Some scholars maintain that there is a full-sized Negroid element in Melanesia which may have a very ancient origin in Asia (Howells, '43, Montagu, '51). Birdsell ('49, p. 6) does not agree with this thesis but states, "I am confident that any so-called Negroid, as opposed to Negritoid type, in New Guinea and Melanesia results from hybridization of Negrito and Carpentarian [Veddoid] elements present throughout much of the archipelago." Whatever the ultimate outcome, it seems legitimate to assume that "some" genetic relationship exists between the African and Melanesian populations.

It has been shown that there is a probable genetic link between Africa, Australasia, and Melanesia. Why then, is the sickle cell gene not present in the last two regions? To answer this, one must determine if the presence of the sickle cell gene in a population is actually indicative of Negro ancestry. At the present time the answer to this question is not satisfactorily known. There are those who maintain that the sickling feature of the erythrocytes cannot be regarded as a peculiarity of the Negro people, and its ethnological significance may have been overrated in the past (Foy *et al.*, '54, '55). In 1952 Lehmann and Cutbush ('52, p. 404) were of the opposite opinion and wrote, "So far as we know, the sickle cell trait and sickle cell anemia have not been reported in people who were not either Africans or members of groups which must be expected to have an admixture of African blood." Lehmann ('53, p. 9), however appears to have shifted his allegiance, for he later wrote, "The trait is not a universal African feature and one may suggest that it entered the continent with Veddian blood well after the African races had established themselves." He also says that the gene cannot be used as a "tracer" of African ancestry as is the case with the R^0 (cDe) chromosome. An interesting suggestion made by von Haimendorf ('54) is apropos here, irrespective of its validity. He believes the introduction of the sickle cell into the blood of the Todas of the Nilgiris Hills of India can perhaps be explained by miscegenation. In 1953 he discovered a hitherto unnoticed system of wife-lending between the Todas and the Mudugas of Pottikal. Certain village headmen of these two groups have traditional friends who visit them and the host is expected to vacate his home, but the wife remains and the visitor assumes the role of the husband. The discovery of this system of hereditary friendships led von Haimendorf ('54, p. 29) to suggest that "the slight incidence of the sickle-cell trait in the blood of Todas can perhaps be explained by such miscegenation . . . and may thus help in the solution of a genetic puzzle." The awareness of such sociological systems may help explain the distribution of a trait among supposedly endogamous groups, but in the present case, it is unknown whether the Mudugas possess the sickle cell gene. (See list of Veddoid tribes examined to date in Lehmann and Cutbush, '52). Thus the verity of von Haimendorf's statement can only be established by further field work. However, such cultural phenomena are too often overlooked by the physical anthropologist unfamiliar with the mores of the people he is studying. Singer ('53, '54), on the other hand, staunchly maintains that the sickle cell gene is found exclusively in Negroes or in persons who have had Negro admixture; in fact, he cites Central Africa as the probable home of the gene. About all one can say at this time is that the presence of the sickle cell in a population is not unequivocal evidence of Negro ancestry. However, irrespective of whether the sickle cell is peculiar to Africans (Singer, '54) or arose among some pre-Veddoid group (Lehmann and Cutbush, '52, Mourant, '52), one must attempt to explain its absence in Melanesia and Australasia because genes from one or the other of these racial elements are supposedly present in this area.

Within the last few years it has been almost conclusively proven that the sickle cell trait (heterozygous condition) has a selective value in a malarious environment (Beet, '46, Choremos *et al.*, '51, Raper, '54, and Allison, '54a, '54b, and '54c). Although this hypothesis is not without its antagonists (notably Foy *et al.*, '55, Moore *et al.*, '54), it is beginning to gain favor, especially since the revealing work by Allison and Raper. These investigators suggest that the heterozygote is favored by a high degree of resistance to malarial parasites, especially with respect to *Plasmodium falciparum*. According to Allison ('54a) *P. falciparum* is the least effective of the malarial parasites since it is not found as frequently as other forms in infected individuals. He further states ('54b, p. 317), "There is little sickle cell south of the Zambesi River because it is not as malarious plus the fact *P. falciparum* is being replaced to some extent by *P. vivax* as the dominant species." It appears that sicklers (heterozygotes) may be naturally resistant to subtertian malaria so that they have a selective advantage over the normal homozygotes in regions where malaria is endemic. In other words, balanced polymorphism may be the explanation for the maintenance of the sickle cell gene. If so, it seems only logical to conclude that the gene had a relatively recent origin, since it is well known that malaria is endemic in most regions of Australasia and Melanesia. By recent I simply mean after the major differentiation and establishment of the populations in those areas, which was undoubtedly sometime during the last half of the fourth glacial period. This suggestion is essentially in agreement with those proposed by Singer ('53), Lehmann ('52), and Mourant ('54).

Another factor which one cannot overlook is genetic drift. If this phenomenon has been operating, present sickling rates in a tribe may not bear much relationship to the frequency of the gene when originally introduced, and individual tribal frequencies would have limited ethnologic significance (Brues, '55). Foy *et al.* ('54) believe that this is the situation which obtains in Africa and that genetic drift, rather than natural selection, is the cause of wide inter-tribal and intra-tribal variability. However, before one too freely entertains the concept of genetic drift to explain all variability, one should recall the criticisms by Glass *et al.* ('52) on such uncritical application of this evolutionary mechanism. With the information now available, balanced polymorphism seems much easier to validate scientifically, although it is quite possible that both phenomena are operating simultaneously on the gene.

CONCLUSION

The previous discussion has attempted to elucidate some of the anthropologic problems concerning the sickle cell gene. It appears that the gene has limited ethnologic application and that its utilization as a "tracer" of Negro ancestry is open to suspicion. The suggestion has been made that the gene never passed eastward of India and that this is a consequence of its relatively recent acquisition in *Homo*. This hypothesis seems even more tenable in view of the data presented here. It is demonstrated that the absence of the gene in Australasia and Melanesia does not substantially alter previous concepts on the origin of these populations.

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MORTALITY OF PERSONS WITH XYLOKETOSURIA

A FOLLOW-UP STUDY OF A RARE METABOLIC ANOMALY

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IT has long been known that xyloketosuria (a condition in which the pentose xyloketose occurs in human urine) is a recessively inherited anomaly which occurs in persons of all age groups (Lasker, Enklewitz and Lasker, '36). Mortality statistics are usually lacking for such anomalies, especially if they are rare or if they can be diagnosed only by special tests. Seventy-two cases of xyloketosuria have been followed, over half of them for 16 years or more. In one case an indirect follow-up of a patient diagnosed as pentosuric in Germany in 1913 was made possible by subsequent independent republication of the same case in South Africa 33 years later (Bloomberg, Alldis and Epstein, '46, and Lasker, '50) and has now been followed for 41 years. In 57 of the cases, I personally tested the urine and established that xyloketose was present. Several pentosurics were tested daily for months at a time, others repeatedly during the long follow-up periods. In 37 of the cases I retested within the last few years, I have found xyloketose present every time and always in a concentration usual for the particular individual. The total follow-up experience is 1043 person-years, an average of 14.5 years per individual.

Of the 72 persons with xyloketosuria, 12 (16.7%) have died. To determine whether this number is significantly more than would have occurred in non-xyloketosuric persons, the data have been compared with life tables. The mid-date between the year of diagnosis and the last year of follow-up was taken for each subject. Those for all the subjects were averaged and the mean falls in 1943. It is therefore appropriate to use data based on the census of 1940 and deaths recorded in the years 1939-1941 (State and Regional Life Tables 1939-1941).

FINDINGS

Table 1 shows the age at which each xyloketosuric case was first identified as one of essential pentosuria and the number of years it was followed. To determine the general white mortality in the United States for persons alive during the years of age corresponding to those of each of the cases, the Life Tables for whites in the United States were consulted. As a control for each case, the entry for the appropriate sex under "Number living at beginning of year of age" for the last age of contact with the pentosuric subject was subtracted from the corresponding entry for the age at first diagnosis, and the difference was divided by the former (State and Regional Life Tables 1939-41, pp. 10-13). The rate of mortality expected in persons between these ages is thus calculated from the table. In the case of persons who died before 1951, follow-up statistics were calculated on the basis of this date, which is the mean termination date of all the other follow-up studies. This is done on the assumption that these individuals, had they lived, could have been studied as easily as the others. The 72 duration-weighted rates were then added to give the total number of deaths expected in a control population of the same age- and sex-distribution. Approximately 8.9 of the 72 (12.4%) would be expected to die in the periods covered.[1]

The question arises whether the difference between the 12 deaths of xyloketosurics and the 8.9 deaths which would have been expected in a corresponding population sample is significant or meaningful. It is neither. Statistically a simple probability test cannot be used because the various cases contribute so unequally to the total experience. But even assuming an "n" of 72, however, the standard error ($\sqrt{\frac{pq}{n}}$) of 16.7% (our percentage mortality) would be $\sqrt{\frac{16.7\% \times 83.3\%}{72}} = \pm 4.4\%$. The difference between the observed and expected mortality ($16.7 - 12.4 = 4.3\%$) would thus be less than the standard error, hence not signifi-

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[1] Three cases which were followed for a year or less have been excluded from consideration because of the shortness of the time they were followed. Had they been included, the mean date of termination of the follow-up periods for surviving individuals would have been less recent and the calculated potential follow-up of those who died would be shorter, hence the calculated expected number of deaths would be somewhat less. On the other hand, the one case in which a subject died within a year of the first diagnosis has been retained in the calculations.

TABLE 1

Follow-up Cases of Xyloketosuria

CASE	AGE PENTOSE IDENTIFIED	FOLLOW-UP PERIOD (YEARS)	YEAR OF LAST CONTACT
Males			
HS	2	3	1955
SB	3	8	1938
LD	3	3	1953
RP	3	4	1955
ME	4	16	1955
AB	4	15	1954
RB	5	41	1954
G	5	7	1953
BC	6	18	1953
MG	6	20	1955
IB	7	8	1955
DF	9	11	1954
HT	10	17	Died 1949
HZ	10	15	1955
RB	11	3	1938
LG	16	2	1938
MB	17	16	1955
L	19	14	1938
K	21	14	1938
JL	22	23	1955
DM	22	14	1952
CS	23	24	1955
LG	24	20	1955
NI	25	19	1955
BL	28	23	1955
HG	28	20	1955
KM	28	11	Died 1948
BR	29	5	1938
LG	30	20	1955
EB	31	20	1955
SS	31	17	1955
NL	33	22	1955
BR	33	1	Died 1934
MD	34	16	1955
JA	35	21	1953
LB	37	12	Died 1944
JD	38	13	Died 1951
BB	39	6	1939
LL	41	23	1954
SB	46	3	1955
ALW	48	30	Died 1938
JS	49	16	Died 1951
F	53	14	1938
AB	57	16	1955
SM	61	9	Died 1946

TABLE 1 (Continued)

Follow-up Cases of Xyloketosuria

CASE	AGE PENTOSE IDENTIFIED	FOLLOW-UP PERIOD (YEARS)	YEAR OF LAST CONTACT
Females			
L	6	3	1953
FD	7	17	1954
RC	8	2	1937
BS	8	6	1955
HD	9	17	1954
HG	9	17	1955
SJ	10	22	1955
JE	11	34	1952
FB	16	3	1938
BM	19	20	1955
EW	19	16	1955
RG	21	16	1955
MKG	23	21	1954
MC	27	21	1953
YZ	29	23	1955
SR	35	15	1952
CJ	37	22	1955
LB	40	12	1953
JG	40	20	1955
AG	41	23	1955
ES	42	3	Died 1939
RY	42	2	1955
RN	51	11	1952
EL	54	12	1945
BL	61	8	Died 1941
FM	67	18	Died 1953
PS	72	6	Died 1944

cant. Actually, the effective "n" is smaller; therefore the standard error is larger, and the difference even less significant. Also, I have used conservative assumptions: 1) For the general population I have used tables which apply to the actual age, that is, presumably about a half year below the average (and hence with lower mortality) than the xyloketosurics, whose ages were recorded as on the last birthday. 2) Although half of the cases came from the State of New York, I have used total United States mortality statistics as a control; the death rates for the State of New York at these ages are appreciably higher. 3) General population mortality statistics were used although the diagnosis of xyloketosuria requires hospital laboratory tests. About 20 of the cases were first discovered in persons hospitalized for some reason unconnected with the sugar in the urine; for example, in one case the patient was in the hospital gravely ill with miliary tuberculosis at the time the xyloketose was identified in the urine, and died of tuberculosis within the year.

DISCUSSION

There is no evidence of an appreciably higher mortality in xyloketosurics than in the general white population of the United States. What little excess of deaths there may be in the former stems from a slight excess of deaths in males 51 years old or less which may be a result of chance (table 2). It may also be seen in table 2 that, in so far as is known, death in xyloketosurics occurs from a range of causes similar to what one might expect in a general population of this age distribution.

TABLE 2

Deaths of Xyloketosurics

CASE	AGE AT DEATH	ASCRIBED CAUSE OF DEATH
Male		
HT	27	Bronchial disease
BR	35	Miliary tuberculosis
KM	39	Polycystic kidney disease
LB	49	Heart disease
JD	51	Coronary thrombosis
JS	65	Coronary thrombosis
SM	70	Unknown
ALW	78	Unknown
Female		
ES	45	Cancer of pancreas
BL	69	Unknown
PS	78	Unknown
FM	85	Unknown

As xyloketosuria is inherited, it may be asked whether the mortality experience can cast any light on the population genetics of this characteristic. As I have said elsewhere (Lasker, '52), the condition seems to be virtually limited to Jews, and the geographical distribution suggests an increase in the gene frequency among them during the last thousand or two thousand years. Such an increase could result from any of three causes: a repeated mutation; some advantage for lower mortality or higher fertility conferred by the gene; or sheer chance factors in a relatively small population. Repeated mutation in a single population would still leave unexplained the reason for a higher mutation rate in one segment of the species but not in other genetically similar peoples; thus in the absence of any direct evidence favoring it, this hypothesis is not helpful.

The present data show no decreased mortality rate for xyloketosurics, but the total mortality experience is too small to permit an answer as to whether this might not be true for the critical years prior to and including the reproductive ages. In any case, for each homozygote, there must be many heterozygotic carriers of xyloketosuria; the mortality and fertility of these would therefore be more important in determining whether there is any "natural selection" favoring a rapid increase in the frequency of the gene for xyloketosuria. Unfortunately, no clinical test is known that will distinguish these carriers. If no factor pointing to natural selection is found, it may well be that in a small isolated population pure chance ("random genetic drift") may have contributed decisively to the present ethnic distribution of this genetic condition.

SUMMARY

In follow-up studies of 72 cases of xyloketosuria, 12 (16.7%) were found to have died. Among white persons of corresponding ages in the United States the death rate is 12.4%. The disparity is not statistically significant. Furthermore, this difference may result from the fact that many of the cases became known only because they were receiving clinical attention for other reasons. Even if the difference were found in much more extensive data—and these would be difficult to collect for such a rare metabolic anomaly—it might be accounted for by the conservative assumptions made in the calculations of the control. As cases are usually found by special clinical tests, some of the xyloketosurics who died may have been detected, and so were included in this study only because they had sought medical advice for illnesses—unrelated to xyloketosuria—which later caused, or contributed toward, their deaths. On the other

hand, the absence of evidence for a lower mortality of xyloketosurics leaves open the question whether favorable mortality and fertility rates (especially among heterozygotic carriers) or sheer chance factors better explain the present distribution of the condition.

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A THEORY OF CULTURAL GENETICS *

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INTRODUCTION

THE core of this presentation may be termed "A Theory of Cultural Genetics" because there are reports in anthropological literature of a variety of human genetic systems which are culturally recognized within each society. Whereas lower forms of animal life ignore or soon forget genetic relationships, human beings maintain their biological relationships through the use of language and records. A particular genetic system is traditionally called a kinship system. The kinship system seldom includes a recognition of all the genetic relationships of the individuals who make up the population, but rather some portion of it which is systematized and known to the people. The people participate upon that basis and thus their daily lives are affected in regard to inheritance, residence after marriage, social relationships of avoidance, reciprocity, respect, joking relations, pre-marital sex relations, and actual marriages. In short, participation in the culture has a direct effect upon the genetics.

The study of human genetics need not be limited to people of a particular time or place. The theory presented herein pertains to the variety of human genetic systems resulting from the reported cultural forms of marriage at many times and places.

Generally speaking marriage is considered the beginning of genetics under consideration. However, no matter which kind of marriage occurs, there is no genetic resultant until children are present. Furthermore if one member of a marriage has a child with a nonmember of that marriage the geneticist is interested in that child and its biological parents rather than the cultural parents. Therefore, it is essential to differentiate between marriage which is cultural, and sexual mating which is genetic.

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* This is an abbreviated presentation of the results of research, experimentation, and testing which began in 1932. Summaries of this paper were presented at the meetings of the American Association of Physical Anthropologists of 1954 and 1955. Several previous publications by the author relate to this subject. (Aginsky, '35A, '35B, '39, '40)

Sororate, levirate, or any other form of marriage, by itself or in combination, when standardized in a population, is of no significance unless children result. When, however, a standardized form of marriage is participated in by the population and is repetitive, then the effects upon the genetics of that population may be of interest to the geneticist.

THE 32 NUCLEAR STRUCTURES

The reported forms of marriage have been brought together without regard to time or space (place). Some of them are: monogamy, polyandry, ploygyny, group, double, exchange, sororate, levirate, mother's brother's wife, husband's sister's son, wife's brother's daughter, father's sister's husband, brother's daughter's husband, cross-cousin, grandparent-grandchild, and brother-sister. Each of these has been reported as being practiced in some societies as the sole form and in others in combinations.

There are 6 basic forms of marriage (see figure 1)

1′P *The Primary* (our own form of sexual mating may serve as an example)

The Secondaries

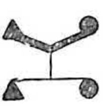
2′S Sororate
3′L Levirate
5′W Wife's brother's daughter
9′H Husband's sister's son
1″E Exchange (Double Primary)

▲ Man
● Woman

Marriage (sexual mating)

Husband and wife (who are parents)

The horizontal line represents siblings (in this case a brother and a sister)


The perpendicular line represents parent-child relationship. (Here we have a brother and sister of the same parents.)

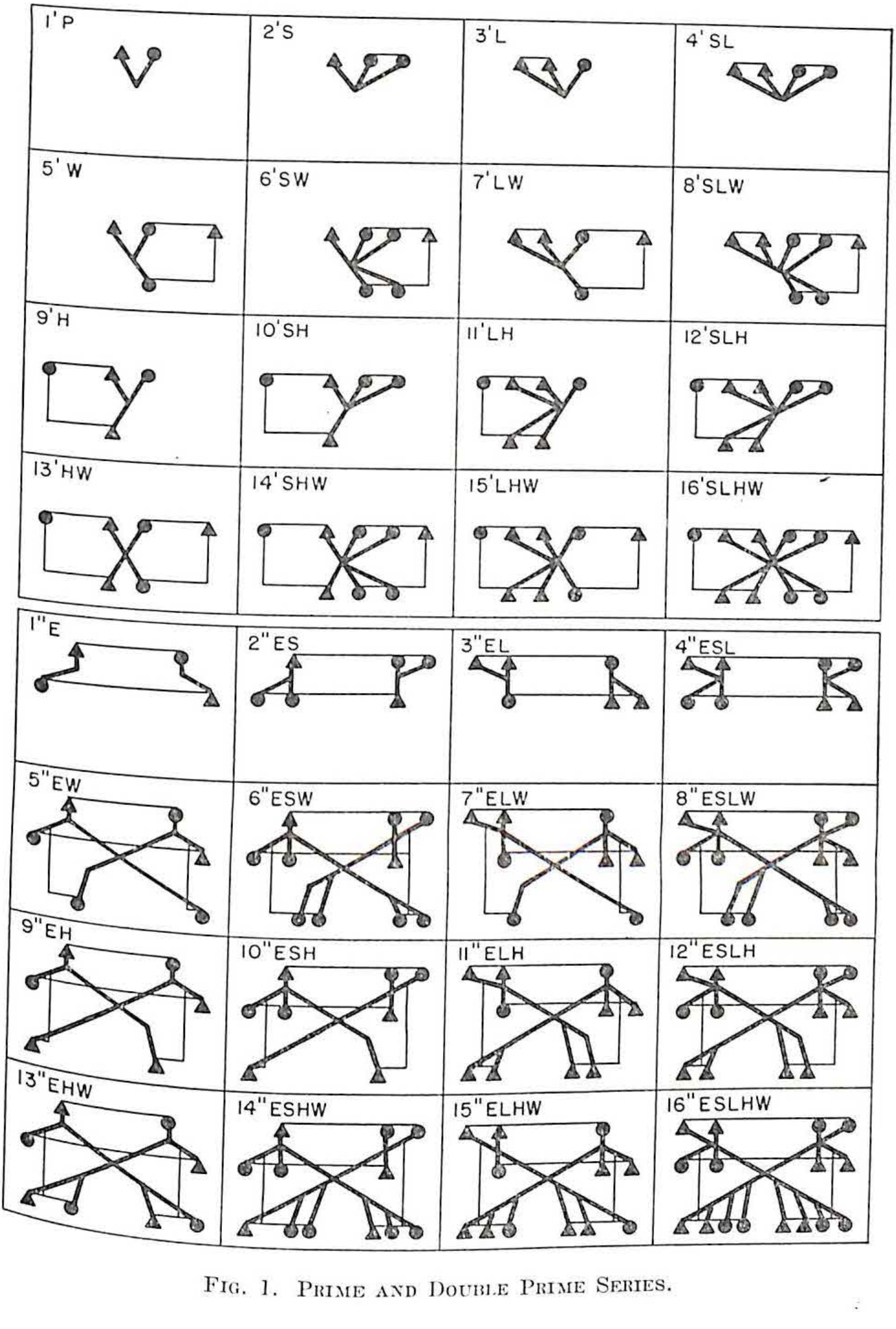


Fig. 1. Prime and Double Prime Series.

Regarding the Primary as universally present we find that when we combine the other 5, one with another, we then have 26 Compounds. The Primary, the 5 Secondaries, and the 26 Compounds add up to a total of 32. These 32 forms of marriage either alone or in combination contain every known type of marriage.

Series. In figure 1 the diagrams for the 32 forms of sexual mating have been presented in a double series. The 16 which include Exchange are doubled forms of the other 16 which do not contain Exchange. Thus there are the Prime Series (1′–16′) and the Double Prime series (1″–16″).

Each of the two major series in turn contains 4 minor series, each of which is represented horizontally in a progression from left to right.

Each of the 32 diagrams of figure 1 shows the nexus of individuals (of the two families) who are involved in each sexual mating and therefore also represents a different nexus of genetic entities.

In a previous study (Aginsky, '35b, pp. 62-64) it was shown that in addition to the Primary and Secondary categories of sexual mating, there is also a tertiary category. It is a combination of plural Secondaries of certain kinds and results in such varieties of sexual mating as cross-cousins, grandparent-grandchild, and brother-sister. In certain cases the Primary disappears and the two or more Secondaries may continue through time without any new Primary occurring. The same is true in certain Compounds which result in Tertiaries. The Tertiary continues and the Secondaries or compounds of Secondaries disappear. The Tertiary then continues without the necessity of any Secondary being practiced.

When any type of sexual mating between related individuals (*e.g.*, grandparent-grandchild, cross-cousins, brother and sister) is considered, it must be borne in mind that there was previously a sexual mating, possibly also between related individuals, which brought each of these related individuals into being.

DERIVATION OF FORMULAE

It is apparent that each of the 32 diagrams consists of a nucleus of individuals who belong to two families. Each of the families is composed of individuals who are either male or female and who belong to the same or different generations.

For the present, *only* the sexual mating between the members of the two families (biological entities) will be considered. In a later section some examples of more than two families will be presented.

Thus we could state: in the Primary, there is present a sexual mating of one man of a family to one woman of another family, but no other sexual mating is present between members of these two families. The other individuals are sexually mated to members of different families.

In Sororate marriage we could say that there is present the sexual mating of one man of a family to the plural women of another family and that no other members of the two families are sexually mated to one another. We can phrase Sororate as the sexual mating of *a single Man* of *Family a* with *plural Women* of *Family b.*

However, unless mention is made of *Generation* one would not know whether one or more than one Generation is involved. To make the statement coincide with the diagram in this respect it is better to say: A *single Man* of *Family a* is sexually mated with *plural Women* of a *single Generation* of *Family b.*

However, this is an inclusive statement and does not exclude other sexual mating between or among other members of both families; therefore it is best to phrase the statement so that it is applicable in the other forms of sexual mating which are to be analysed as we proceed. Thus the total statement is:

> A *single Man* of a *single Generation* (but *zero Woman*) of *Family a* is involved with *plural Women* of a *single Generation* (but *zero Man*) of *Family b.*

This can be abbreviated into a formula:

$$S = (MsGs)\,(WzGz)\,Fa \vee (WpGs)\,(MzGz)\,Fb$$

M	represents	Man
W	"	Woman (elsewhere W sometimes stands for mating with Wife's brother's daughter)
G	"	Generation
F	"	Family (biological entity)
a or b	"	particular families
z	"	zero or none
s	"	single or one
p	"	plural, or more than one
$\vee$	"	sexual mating (marriage) between or among

Upon analysis we find that every form of sexual mating contains the same factors. These factors we shall consider invariants. It has also been found that these invariant factors are found in a universal interrelationship. That is, all of the factors are always present. The quantity of each factor may vary.

The universal equation. Each of the 32 diagrams represents the nexus of the male(s) and female(s) of the two biological entities who are directly involved in the particular variety of sexual mating. Each nexus results in what we shall term nucleus. Each diagram shows the number of individuals involved from each family. If we delete the quantitative differences of: s, p, and z, and the indications of Family, Fa and Fb, we then have as that which is common to all 32 formulae the following:

$$(MG)(WG) \vee (WG)(MG).$$

This formula indicates only that there is present a sexual mating relationship between and among the members of two biological entities without any indication of quantity. It is a universal formula.

Sex may be thought of as a force which is manifested in, for example, sexual intercourse with animals, with inanimate objects, with self, with members of the same sex, and with members of the opposite sex. It is this last category of manifestations—heterosexuality, upon which we have focused our investigation and analysis. Of all the manifestations in this category we are here concerned only with marital sexual relations which result in children, and thus "the family."

(We shall use "Λ" to designate the force Sex since "S" has been preempted for Sororate and "s" for single.) The universal equation is $\Lambda = (MG)(WG) \vee (WG)(MG)$.

If we give Λ some definite quantity we find that there is a corresponding quantity manifested in the right hand side of the equation, *e. g.*:

Universal equation	$\Lambda = (MG)\ (WG) \vee (WG)\ (MG)$
1′ Primary	$P = (MsGs)(WzGz)Fa \vee (WsGs)(MzGz)Fb$
2′ P plus Sororate	$S = (MsGs)(WzGz)Fa \vee (WpGs)(MzGz)Fb$
3′ P plus Levirate	$L = (MpGs)(WzGz)Fa \vee (WsGs)(MzGz)Fb$
4′ P plus S plus L	$SL = (MpGs)(WzGz)Fa \vee (WpGs)(MzGz)Fb$

We can tentatively conclude that each nucleus is the result of sexual mating and varies proportionately to the quantity of Λ.

Table 1 contains the 32 formulae. It is divided into the Prime and Double Primes series, each of which is subdivided into 4 minor series.

TABLE 1

Prime and Double Prime Series 32 Forms of Sexual Mating

1′	P	= (MsGs) (WzGz) Fa ∨ (WsGs) (MzGz) Fb	Mono-generational males and females
2′	PS	= (MsGs) (WzGz) Fa ∨ (WpGs) (MzGz) Fb	
3′	P L	= (MpGs) (WzGz) Fa ∨ (WsGs) (MzGz) Fb	
4′	PSL	= (MpGs) (WzGz) Fa ∨ (WpGs) (MzGz) Fb	
5′	P W	= (MsGs) (WzGz) Fa ∨ (WsGp) (MzGz) Fb	Mono-gen: males Poly-gen: females
6′	PS W	= (MsGs) (WzGz) Fa ∨ (WpGp) (MzGz) Fb	
7′	P LW	= (MpGs) (WzGz) Fa ∨ (WsGp) (MzGz) Fb	
8′	PSLW	= (MpGs) (WzGz) Fa ∨ (WpGp) (MzGz) Fb	
9′	P H	= (MsGp) (WzGz) Fa ∨ (WsGs) (MzGz) Fb	Poly-gen: males Mono-gen: females
10′	PS H	= (MsGp) (WzGz) Fa ∨ (WpGs) (MzGz) Fb	
11′	PL H	= (MpGp) (WzGz) Fa ∨ (WsGs) (MzGz) Fb	
12′	PSLH	= (MpGp) (WzGz) Fa ∨ (WpGs) (MzGz) Fb	
13′	P WH	= (MsGp) (WzGz) Fa ∨ (WsGp) (MzGz) Fb	Poly-gen: males Poly-gen: females
14′	PSWH	= (MsGp) (WzGz) Fa ∨ (WpGp) (MzGz) Fb	
15′	P LWH	= (MpGp) (WzGz) Fa ∨ (WsGp) (MzGz) Fb	
16′	PSLWH	= (MpGp) (WzGz) Fa ∨ (WpGp) (MzGz) Fb	
1″	P E	= (MsGs) (WsGs) Fa ∨ (WsGs) (MsGs) Fb	Mono-gen: males Mono-gen: females
2″	PS E	= (MsGs) (WpGs) Fa ∨ (WpGs) (MsGs) Fb	
3″	P L E	= (MpGs) (WsGs) Fa ∨ (WsGs) (MpGs) Fb	
4″	PSL E	= (MpGs) (WpGs) Fa ∨ (WpGs) (MpGs) Fb	
5″	P W E	= (MsGs) (WsGp) Fa ∨ (WsGp) (MsGs) Fb	Mono-gen: males Poly-gen: females
6″	PS W E	= (MsGs) (WpGp) Fa ∨ (WpGp) (MsGs) Fb	
7″	P LW E	= (MpGs) (WsGp) Fa ∨ (WsGp) (MpGs) Fb	
8″	PSLW E	= (MpGs) (WpGp) Fa ∨ (WpGp) (MpGs) Fb	
9″	P HE	= (MsGp) (WsGs) Fa ∨ (WsGs) (MsGp) Fb	Poly-gen: males Mono-gen: females
10″	PS HE	= (MsGp) (WpGs) Fa ∨ (WpGs) (MsGp) Fb	
11″	P L HE	= (MpGp) (WsGs) Fa ∨ (WsGs) (MpGp) Fb	
12″	PSL HE	= (MpGp) (WpGs) Fa ∨ (WpGs) (MpGp) Fb	
13″	P WHE	= (MsGp) (WsGp) Fa ∨ (WsGp) (MsGp) Fb	Poly-gen: males Poly-gen: females
14″	PS WHE	= (MsGp) (WpGp) Fa ∨ (WpGp) (MsGp) Fb	
15″	P LWHE	= (MpGp) (WsGp) Fa ∨ (WsGp) (MpGp) Fb	
16″	PSLWHE	= (MpGp) (WpGp) Fa ∨ (WpGp) (MpGp) Fb	

In brother-sister marriage (1MG1) Fa ∨ (1WG1) Fa, the G1 represents the identical generation for both the brother and the sister. It is also possible to represent the marriage of any other two or more relatives.

In a later section we shall derive finite quantities and develop our formulae further. This will be done for both M-W and for G.

Substitution of Quantities for s *and* p. It is rather simple to substitute quantities for s and p, for example in a case where two men of Family a in the first generation are both married to one woman of one generation of Family b (fraternal polyandry) that nexus may be indicated as follows: (2MG1) Fa ∨ (1WG1) Fb. The formula (4MG1) (2WG2) Fa ∨ (2WG2) (IMG1) Fb, indicates that 4 men of the first generation of Family a are married to 2 women of the second generation of Family b and that 2 women of the second generation of Family a are married to one man of the first generation of Family b.

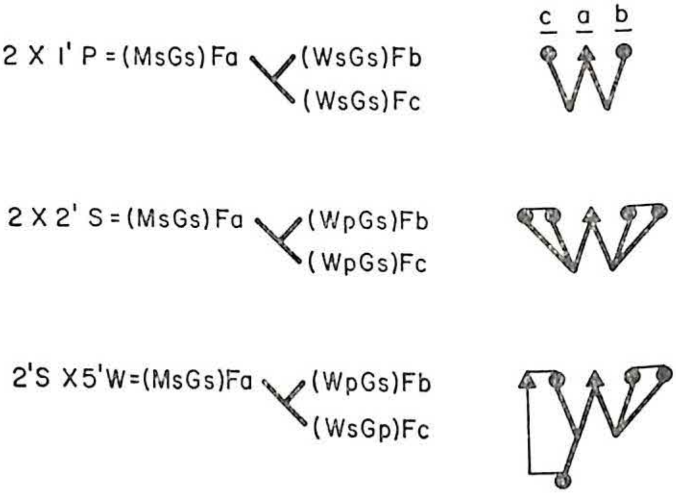


FIG. 2. SOME EXAMPLES OF FORMULAE REPRESENTING SEXUAL MATING OF INDIVIDUALS FROM MORE THAN TWO FAMILIES.

(2MG1)Fa
(1MG2)Fa mated with (4WG1)Fb or (2MG1)(1MG2)Fa ∨ (4WG1)Fb

Both of these indicate the same nexus, namely, that 2 men of generation 1 and one man of generation 2 of Family a are married to 4 women of generation 1 of Family b. For further examples see figure 2.

PROGRESSIONS

Upon analysis of figure 1 we find that the progressions in the minor series are not quantitatively uniform. For example, 1′–4′ (P, S, L, SL), starts with Primary, goes to Sororate, then Levirate, and then Sororate + Levirate. By examination we find that Sororate (2′) and Levirate (3′) are opposite forms of sexual mating and appear to be of the same value quantitatively. Therefore, the progression should be from 1′ (Primary) to 2′ or 3′ (Sororate or Levirate) which should be on the same level as each other and then to 4′, which is the total of 2′ and 3′. Thus the progression is: 1′ (2′–3′) and 4′, as represented in figure 3. Upon examination we find that the minor series 9–12 is the same as 1–4. Numbers 10 and 11 are of the same quantity and are opposites. The series 5–8 is opposite the series 9–12 but of the same value. Therefore the diagram should be arranged to indicate that these values are quantitatively parallel. The final minor series, 13–16, is greater in quantity than those which precede it but it is similar to them in that 14 and 15 are opposites.

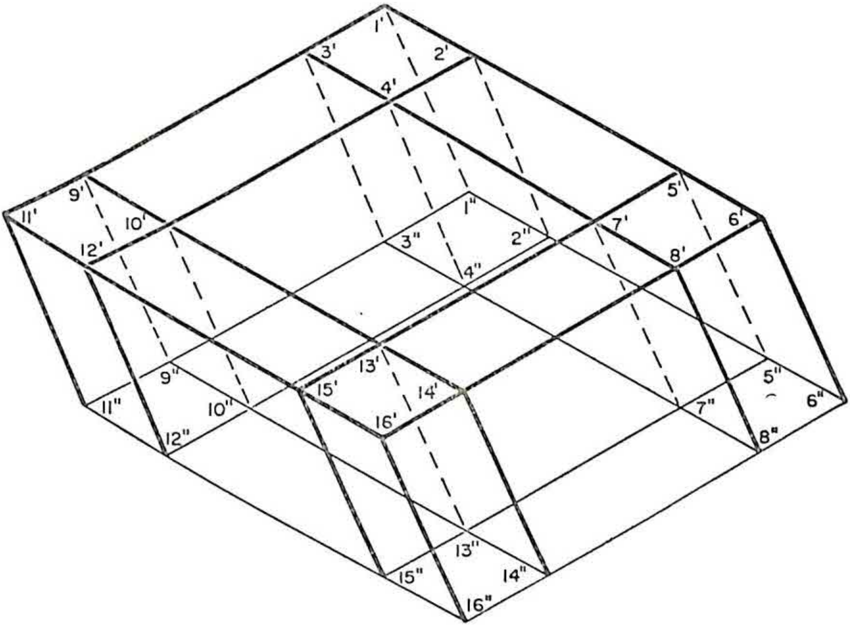


FIG. 3. PROGRESSIONS.

Therefore, a table of progressions for each of the Prime and Double

Prime series could bring together the 4 progressions. Each level should be considered, tentatively, as containing those of the same quantity. The nuclear structures of the Double Prime series, are also presented in an identical 4-fold diamond pattern. It appears that in each of the 4-fold diamonds quantitative change would most easily follow along the diagonals. The line of change could easily branch at a right angle at any point and proceed along a new diagonal or it could reverse directions at any point. However, it would appear to require a greater amount of change for the direction to be at a 45° angle to these or to skip in other directions.

When we superimpose one diamond upon the other we can see that the change from one series to the other does not require too great an amount of change. The quantification of amount of change from one form of sexual mating to another along the diagonals, horizontals, and between the two major series, that is, the change from any one form of sexual mating to another of the 32, would require additional study, however.

As the quantity of A is increased or decreased there is a proportionate change in the nuclear structure of sexual mating. Therefore, a more exact table of progressions would have to be presented as a more complex three-dimensional form. The exact form of that "cube" would depend on the quantification of the 32 forms.

If the 32 contain all of the reported forms of sexual mating and in addition others which have not been reported as yet, it may very well be that we have succeeded in indicating a table of mathematical progressions which has some relation to actual changes in mating patterns.

CONSTANTS AND PROGRESSIONS

When we examine the diamond shaped chart (figure 3) and follow the diagonals, from top left to bottom right we find that for the Prime series:

In 1, 2, 5, 6, the quantity of the male factor MsGs is constant, while the quantity of the female factor increases progressively from WsGs to WpGs to WsGp to WpGp.

In 3, 4, 7, 8, the quantity of the male factor is MpGs and remains constant while the quantity of the female factor increases progressively from WsGs to WpGs to WsGp to WpGp.

In 9, 10, 13, 14, the quantity of the male factor is MsGp and remains constant while the quantity of the female factor increases progressively from WsGs to WpGs to WsGp to WpGp.

In 11, 12, 15, 16, the quantity of the male factor is MpGp and remains constant while the quantity of the female factor increases progressively from WsGs to WpGs to WsGp to WpGp.

On the other hand, if we follow the diagonals in the diamond from top right to bottom left the female factor remains constant in each of the diagonals while the male factor increases progressively in each step.

We have here tables of progressions which indicate, not an infinite continuous series, but rather a finite mathematical progression based upon the quantities of the invariant factors of Gender (Man and Woman), and Generation. The quantities are s and p for both Man and Woman and Generation of each biological entity (family).

We now can see more easily 1) that the same factors and the interrelation of those factors are always present and 2) that a change in quantity of the force Sex (A) results in a corresponding change in the form of sexual mating and the resulting nuclear structure. It can also more easily be seen that the nuclear structures of sexual mating are directly proportional to the quantity of A.

PROBABILITY

Upon examination of the diagrams of figure 1, we find that the Primary is present in all 32 forms of sexual mating, while each of the 5 Secondaries occurs in every possible combination. Thus, each Secondary is found as an element in 16 of the forms, once by itself and 15 times in Compounds with one or more of the other Secondaries.

The forms in which different elements occur are shown in table 2. If each one of the 32 forms of sexual mating were found with equal frequency, this table of probabilities would show the frequencies of the elements. Excluding all other matters one would expect to find Sororate (or L, or W, or H, or E) in half of the matings, and in 15/32 of the cases it would be found as part of a Compound combined with other Secondaries. However, in order to achieve any worthwhile degree of correlation with actual cases, it would be essential to understand additional factors which are involved in determining the form of sexual mating present in any actual case.

An hypothesis. In populations which are evenly divided between males and females of sexual mating age it seems, although there may be

TABLE 2

Probabilities of Various Elements in the 32 Forms of Sexual Mating

	Total	Prime series																Double Prime series															
Element	No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
P	32	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
S	16		0		0		0		0		0		0		0		0		0		0		0		0		0		0		0		0
L	16			0	0			0	0			0	0			0	0			0	0			0	0			0	0			0	0
SL	8				0				0				0				0				0				0				0				0
W	16					0	0	0	0					0	0	0	0					0	0	0	0					0	0	0	0
S W	8						0		0						0		0						0		0						0		0
LW	8							0	0							0	0							0	0							0	0
SLW	4								0								0								0								0
H	16									0	0	0	0	0	0	0	0									0	0	0	0	0	0	0	0
S H	8										0		0		0		0										0		0		0		0
L H	8											0	0			0	0											0	0			0	0
SL H	4												0				0												0				0
WH	8													0	0	0	0													0	0	0	0
S WH	4														0		0														0		0
LWH	4															0	0															0	0
SLWH	2																0																0
E	16																	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
E S	8																		0		0		0		0		0		0		0		0
E L	8																			0	0			0	0			0	0			0	0
E SL	4																				0				0				0				0
E W	8																					0	0	0	0					0	0	0	0
E S W	4																						0		0						0		0
E LW	4																							0	0							0	0
E SLW	2																								0								0
E H	8																									0	0	0	0	0	0	0	0
E S H	4																										0		0		0		0
E L H	4																											0	0			0	0
E SL H	2																												0				0
E WH	4																													0	0	0	0
E S WH	2																														0		0
E LWH	2																															0	0
E SLWH	1																																0

a variety of forms of sexual mating in practice, that the forms would be in balance. The relationships between forms indicated in the diamond of figure 3 may be of aid in this matter.

One might expect to find, for example,

2′ and 3′ being practiced simultaneously or both together as 4′, or
5′ and 9′ " " " " " " " 13′, or
6′ and 11′ " " " " " " " 16′, or
all 16 " " " " all " " 16′, and
similarly for the Double Prime series.

However, in populations where the ratio of males and females is not equal one might expect to find different forms practiced. With a preponderance of males, L and/or H might be found with no other form, or in combination with other forms depending upon the ages, sex ratio, size of the population etc.

THE RESULTANTS AND QUANTIFICATION

The previous diagrams represent only the nuclei of the 32 varieties of sexual mating. We shall now present the resultants, *i. e.*, the kinship systems, of the Primary and the 5 Secondaries, and also 4′ (2′ + 3′) as an example of a Compound.

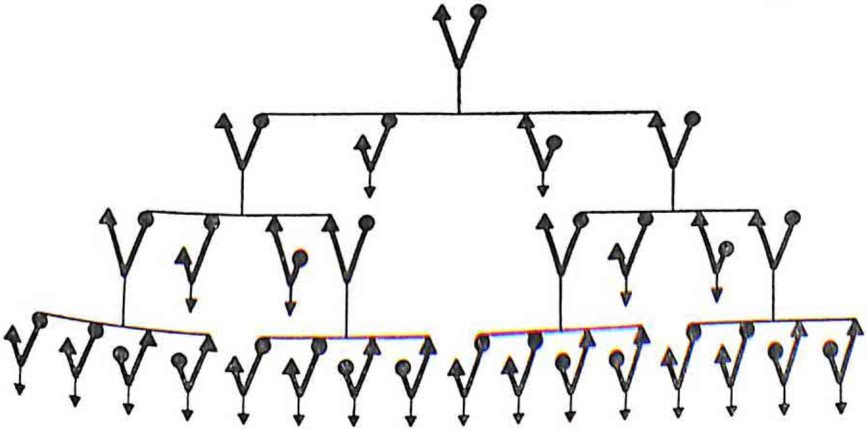


FIG. 4. 1′P. GENETIC AND CULTURAL.

The Primary diagram is presented first (figure 4) and represents both the genetic and the cultural. It is a genetic chart with 4 children indicated for each married female. Since the number of children is proportional to the number of female parents, 4 children will be shown for each female parent in the diagrams which follow.

Figures 5, 7, 9, 12 and 14 are presented as the "cultural" result of each form of sexual mating and marriage wherein those children who have at least one parent in common are placed in the same sibling group. However, an example of the genetic results of each sexual mating form is illustrated by separate diagrams (figures 6, 8, 10, 13 and 15), wherein the genetic lines for each married individual are indicated and the sibling relationships of each sibling group are indicated by breaks in the horizontal line.

By comparing the kinship systems with one another, one may note the differences in the nuclear family structures, constellations of relatives, number of relationships, number of individuals in each relationship, and resultant genetic relationships.

The kinship systems which are presented diagramatically should be considered as "pure" laboratory constructs. Actual kinship systems may be compared with them. Attention is directed to the repetitive pattern within each diagram. Due to the limitation of space, arrows have been used to indicate repetitive descending groups.

One may calculate the quantity in any generation by multiplying the number in one group of a descending line by the number of descending lines. For example, in 1′, there are 4 descending lines. When we count the number of new families involved—marriage groups, number of descendants, number of strangers, etc.—in one descending line in the second generation we find: 4 families, 4 descendants, 4 strangers or a total of 8. When we multiply these figures by 4 descending lines we then have 16, 16, 16, and 32.

Table 3 presents the numbers for each of 1′, 2′, 3′, and 4′. A comparison indicates that there is a difference of genetic "spread" or "contraction."

Before going on to 5′ and 9′ where plural generations are involved we shall discuss the quantitative aspect involved in the numbers of men, women and generations.

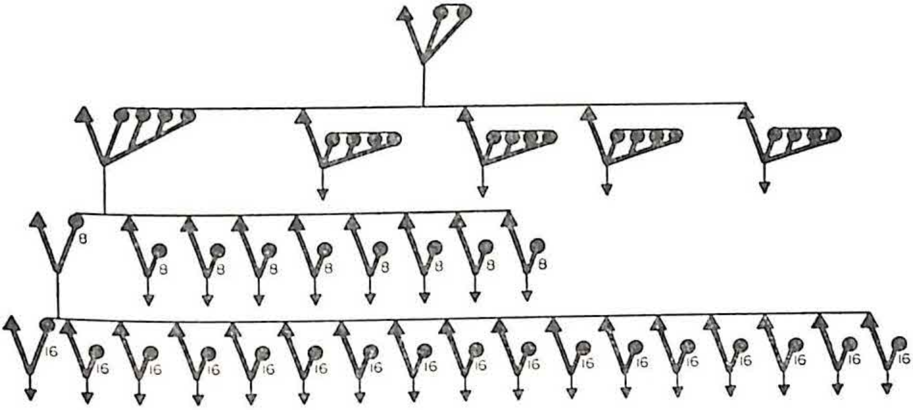


FIG. 5. 2'S. CULTURAL.

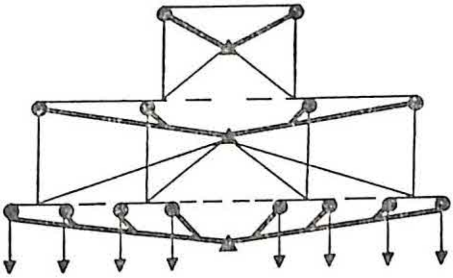


FIG. 6. 2'S. GENETIC.

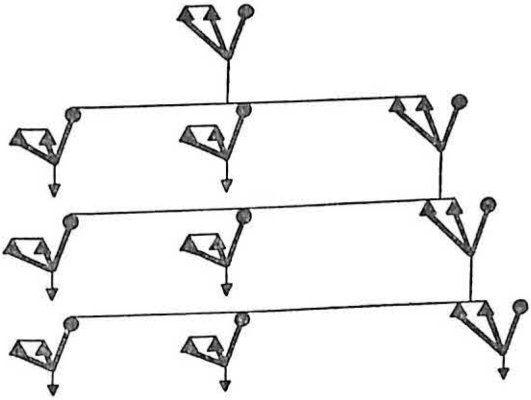


FIG. 7. 3'L. CULTURAL.

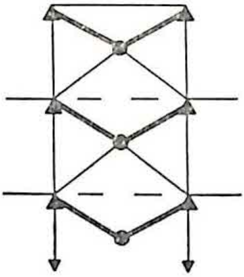


FIG. 8. 3'L. GENETIC.

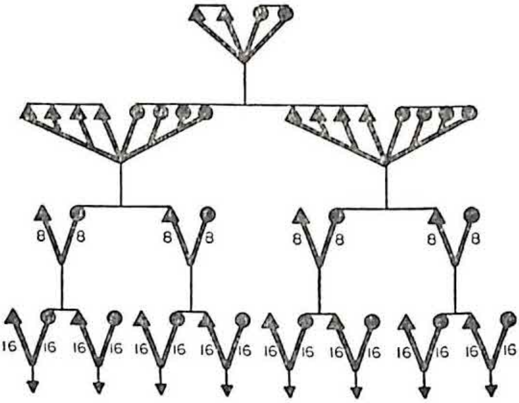


FIG. 9. 4'SL. CULTURAL.

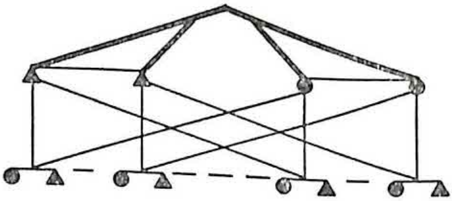


FIG. 10. 4'SL. GENETIC.

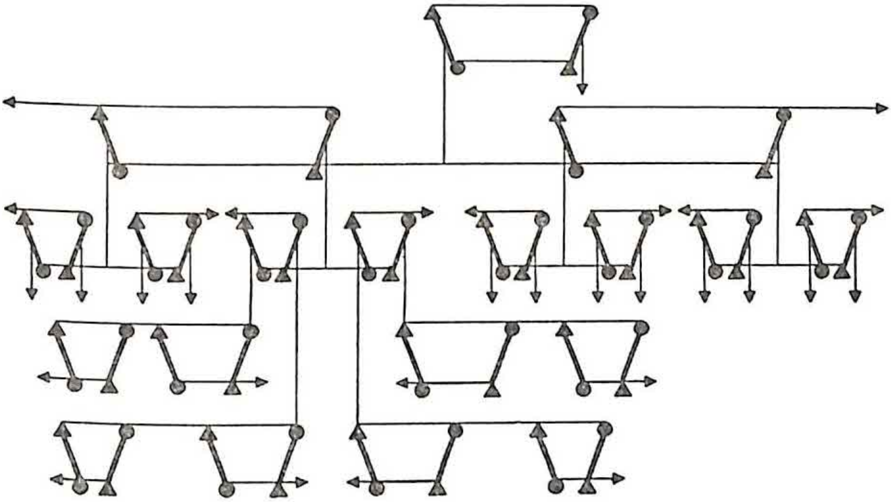


FIG. 11. 1″E. GENETIC AND CULTURAL.

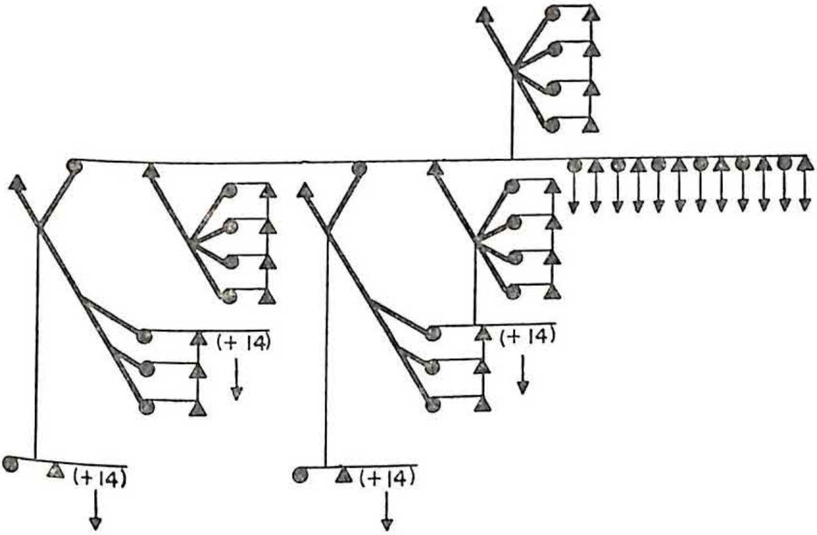


FIG. 12. 5′W. CULTURAL. Since a man may impregnate for 60 years he can mate with women of 4 generations. Note that, to a woman, her own children and her brother's daughter's children have the same father (her own husband); to a man, his sister's children and his daughter's children have the same father.

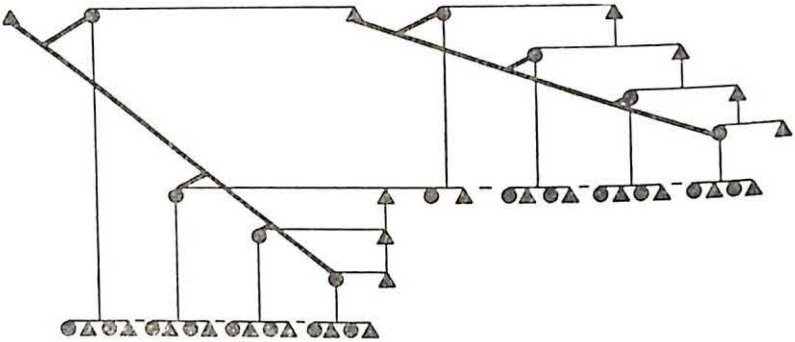


FIG. 13. 5′W. GENETIC.

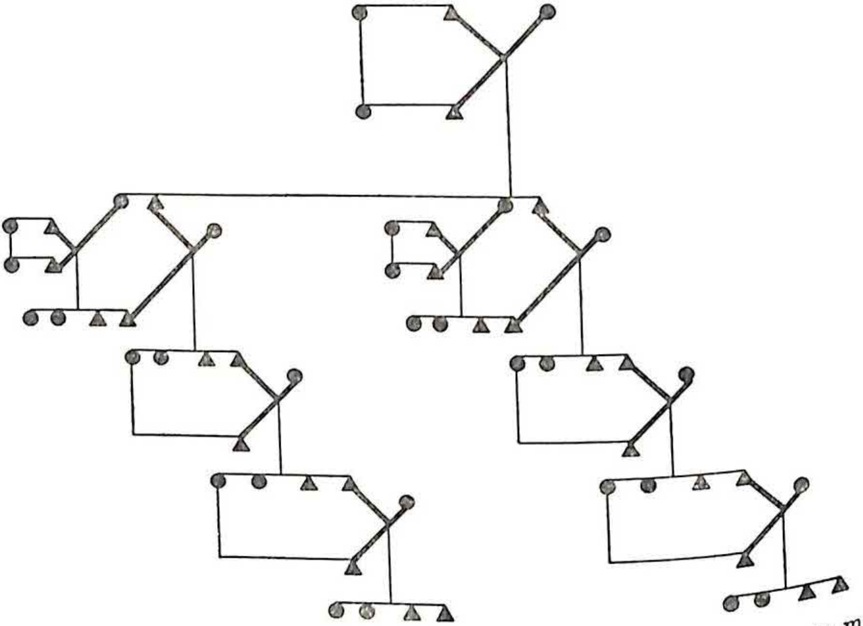


FIG. 14. 9′H. CULTURAL. Since a woman can bear for 30 years she can mate with men of two generations. Note that, to a man, his own children and his sister's grandchildren have the same mother (his own wife); to a woman her brother's children and her son's children have the same mother.

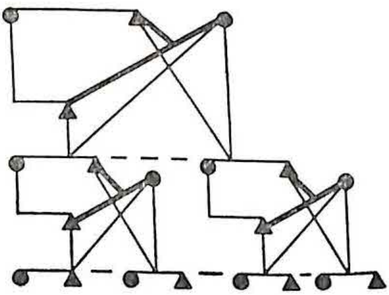


FIG. 15. 9′H. GENETIC.

TABLE 3

Quantitative Comparison 1′, 2′, 3′, and 4′

	1′				*2′*				*3′*				*4′*			
G	F	D	ST	T	F	D	ST	T	F	D	ST	T	F	D	ST	T
1	4	4	4	8	5	8	17	25	3	4	5	9	2	8	8	16
2	16	16	16	32	45	80	325	405	9	12	15	27	4	32	32	64
3	64	64	64	128	765	1,440	11,565	13,005	27	36	45	81	8	128	128	256
TT	84	84	84	168	815	1,528	11,907	13,435	39	52	65	117	16	168	168	336

G	Generation
F	new families involved and sexual mating groups
D	number of descendants
ST	number of strangers involved
T	total strangers and descendants
TT	total of all in third generation

SUBSTITUTION OF QUANTITIES FOR THE SYMBOLS M, W, G, AND THE VALUES s AND p

The quantities below have been chosen arbitrarily for easy computation and to indicate a method by which quantification may be accomplished in the formulae:

Initial child-bearing by women 15 years of age; initial impregnation by men 15 years of age. (Therefore each generation amounts to 15 years.) Terminal child-bearing by women 44 years of age. (Thus a woman bears children for 30 years.) Terminal impregnation by men 74 years of age. (Thus a man impregnates for 60 years.) Therefore the value of WG is 2 (Generations) and the value of MG is 4 (Generations) when plural generations are involved.

Upon this basis in 5′ W the man may marry the W in 4 generations of his wife's family, while in 9′H the woman may marry the M in two generations of her husband's family. When W and H are found in the same compound (13′, 14′, 15′, 16′), the result is that a perpetual system comes about. We shall indicate this as 2+ for men and 5+ for women.

It can be seen that where Gs is involved, *e. g.*, MsGs ∨ WsGs (and this condition obtains for the lifetime of the man and the woman) the result is determined by the factor W which limits the factor M, and arbitrarily using 4 children for each woman there will be but 4 children. In 2′ S, MsGs ∨ WpGs, there will be 8 children and again the result is limited by the factor W.

However, where Gp is the quantity the matter is more complicated as can be seen in table 4. Here quantities have been substituted for the symbols in each of the formulae in the Prime series. Across the top of the table are listed both s and p for W, M, and G.

Since there are different combinations of parents in each of the 16 forms of sexual mating in the Prime series (and also in the Double Prime series) the genetic composition of the descendants will also be different in each of the 16. Although the total of Qf and Qm, that is parents, may be the same there is a different genetic resultant. For example in 3′L there are 2 fathers who are brothers as compared with 9′H where there are 2 fathers who are mother's brother and sister's son.

Should we apply the quantities presented in this section to cultural genetics we find that at the end of 180 years, counting generations through the first born of the first born upon the basis of 15 years per generation there would be a total of 12 generations preceding the last individual. If the count is through the last born female of the last born female and using 30 years as the gauge there would be but 6 generations, and if the count were through the last born male of the last born male with the gauge being 60 years there would be but 3 generations. At the end of 180 years, although all 3 individuals (first born of first born, last born female of last born female, and last born male of last born male) would be born in the same year, each would have an entirely different quantitative generational genetic history. In short, the genetic effects of males and females in the population would vary considerably.

TABLE 4

The Substitution of Quantities for Symbols in the Prime series

		Q of f				Tf	Q of m				Tm	Qch	T in Nu F	Tfm
		Ms	Mp	Gs	Gp		Ws	Wp	Gs	Gp				
1′	P	1		1		1	1		1		1	4	6	2
2′	S	1		1		1		2	1		2	8	11	3
3′	L		2	1		2	1		1		1	4	7	3
4′	SL		2	1		2		2	1		2	8	12	4
5′	W	1		1		1	1			4	4	16	21	5
6′	SW	1		1		1		2		4	8	32	41	9
7′	LW		2	1		2	1			4	4	16	22	6
8′	SLW		2	1		2		2		4	8	32	42	10
9′	H	1			2	2	1		1		1	4	7	3
10′	SH	1			2	2		2	1		2	8	12	4
11′	LH		2		2	4	1		1		1	4	9	5
12′	SLH		2		2	4		2	1		2	8	14	6
13′	HW	1			2+	2+	1			5+	5+	20+	27+	7+
14′	SHW	1			2+	2+		2		5+	10+	40+	52+	12+
15′	LHW		2		2+	4+	1			5+	5+	20+	29+	9+
16′	SLHW		2		2+	4+		2		5+	10+	40+	54+	14+

Q of f equals the number of fathers
Tf " total fathers
Q of m " the number of mothers
Tm " total mothers
Q of ch " quantity of children
T in Nu F means Total in Nuclear Family and is the addition of Tf, Tm, and Qch. Tfm is the total of Tf and Tm and also represents the quantity of genetic entities.

By now it is clear that the progressions illustrated in figure 3 must be redrawn on the basis of 4 generations for males and 2 generations for females, (figs. 12-15).

THE WIDER FOCUS

As we construct larger nuclear sexual mating groups, we are simultaneously enlarging the sexual mating groups for all the members of the two sibling groups. Thus, the size and composition of each spouse's sibling's affinal family varies in each of the 7 cases, the variation is proportional to the form of sexual mating, and is correlated with the size and composition of the nuclear family. Figures 16-20 illustrate this point.

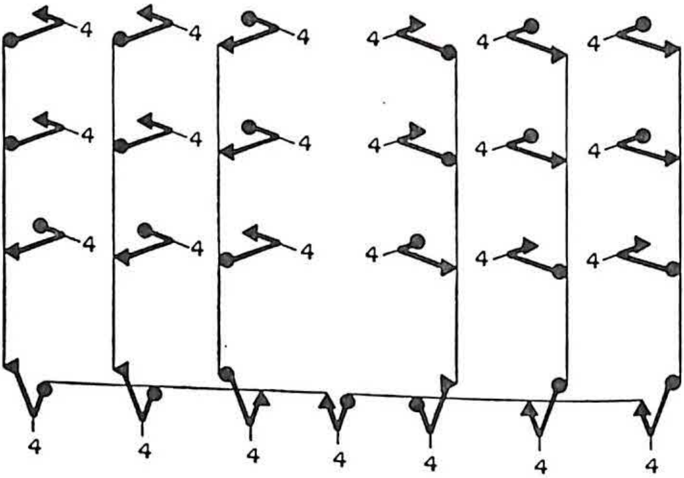


FIG. 16. MODEL USING 1′P. There are 25 nuclear families present, each consisting of 6 members. There are 25 fathers, 25 mothers and 100 children totalling 150 persons.

For the sake of simplicity we shall present the diagramatic results only for 1′, 2′, 3′, 4′ and 1″. The numeral in the box indicates the number of children of the sexual mating group.

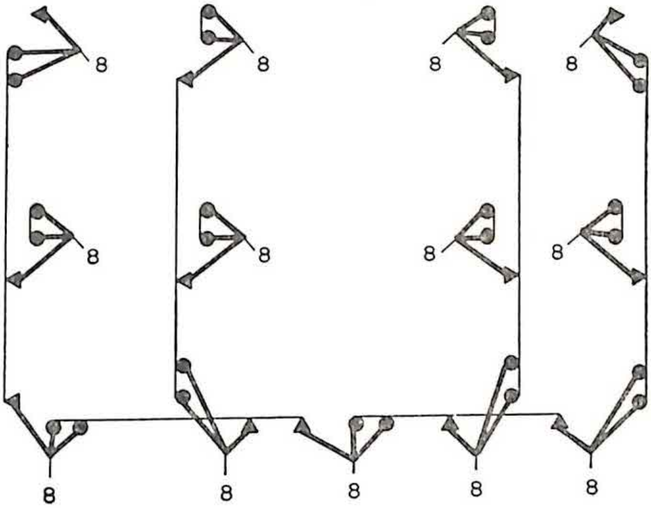


FIG. 17. MODEL USING 2'S. There are 13 nuclear families present, each consisting of 11 members. There are 13 fathers, 26 mothers and 104 children totalling 143 persons.

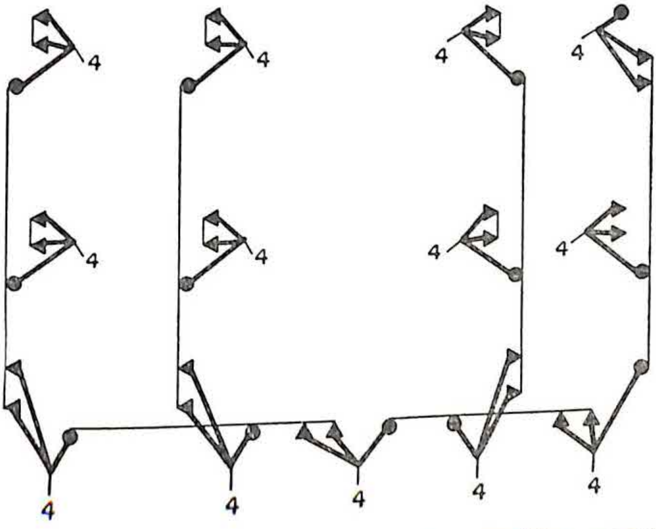


FIG. 18. MODEL USING 3'L. There are 13 nuclear families present, each consisting of 7 members. There are 26 fathers, 13 mothers and 52 children totalling 91 persons.

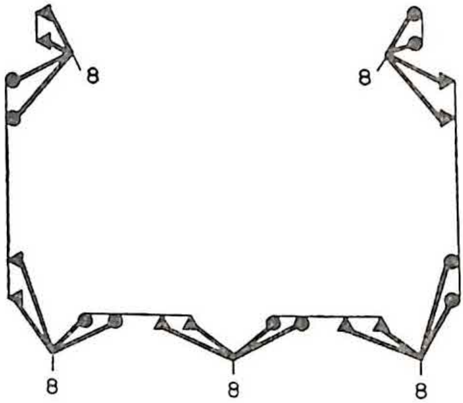


FIG. 19. MODEL USING 4′SL. There are 5 nuclear families present, each consisting of 12 members. There are 10 fathers, 10 mothers and 40 children totalling 60 persons.

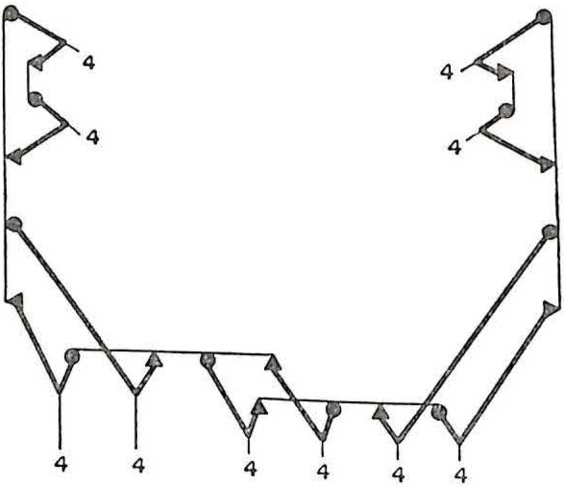


FIG. 20. MODEL USING 1″E. There are 10 nuclear families present, each consisting of 6 members (actually 5 double families of 12 each). There are 10 fathers, 10 mothers and 40 children totalling 60 persons.

Within the present focus the differentials of factors M and W even out to some extent and, as diagramed, it appears that the greater the quantity of the force Sex (A) the smaller the total number of individuals involved.

TABLE 5

Comparative Table

	1′	*2′*	*3′*	*4′*	*1″*
1. Number of individuals in each sexual mating group	2	3	3	4	2
2. Number of children in each nuclear family	4	8	4	8	4
3. (no. 1 + no. 2) total individuals in nuclear family	6	11	7	12	6
4. Number of sexual mating groups	25	13	13	5	10
5. (no. 1 × no. 4) total individuals-sexual mating groups	50	39	39	20	20
6. (no. 2 × no. 4) total children	100	104	52	40	40
7. (no. 5 + no. 6) total individuals	150	143	91	60	60

The greater the amount of A, the larger the particular sexual mating group, the fewer the number of separate sexual mating groups and nuclear families, the smaller the total quantity of individuals involved, and the smaller the population.

Therefore the smaller the total population the larger the sexual mating group would tend to be. In short, one would anticipate a correlation between the total size of the population and the form of sexual mating.

RELATIONSHIPS, AFFINAL AND CONSANGUINEAL, COMPOSITION AND QUANTITY

In the Primary 1′, the maximum number of relationships remain in the affinal families since only one individual has been taken from each of the two families to construct the new nuclear family, *i. e.*, husband and wife. To the man, and also to the woman, of this sexual mating group, each affinal family consists of the remaining individuals of various relationships. For the sake of simplicity we shall include only the members of the nuclear families. Thus we shall utilize only the members of the sexual mating groups and their children.

Table 6 contains the relationships which are present or absent and the quantity of individuals in each relationship for 1′, 2′, 3′, 4′ and 1″. Both the man (column II) and the woman (column III) relate to the identical individuals. Thus there are always at least two relationships present in regard to each individual, one for the man and one for the woman except for husband and wife. (Column I is the total of II and III.)

Column I contains the quantities of relationships for 1′. The nuclear family of 1′ contains 6 relationships. Each succeeding line contains 8 relationships. At the bottom of the column we find the total 6/32. The 6 represents the number of relationships in the nuclear family while the 32 represents the total relationships in the combined affinal and consanguineal families.

When we follow across the grand total line we find, under IV, the quantities of relationships is repeated under 1′ and then the grand totals for 2′, 3′, 4′ and 1″. The line above entitled "Individuals" contains the total of A and the grand totals of BCDE. Thus under IV 1′ is found 6/36. The 6 represents the number of individuals in the nuclear family while the 36 represents the total number of individuals in the combined affinal and consanguineal families.

In 2′, the man, being married to his wife's sister, does not have a female affinal nuclear family and therefore does not have some of the relationships present for the man as in 1′, namely, wife's sister, wife's sister's husband, wife's sister's son and daughter. The woman does not have a female consanguineal nuclear family. Here her sister is her co-wife and her sister's children are her husband's children and are members of her own nuclear family. However the man now has:

4 wife's brother's wives instead of 2
8 wife's brother's sons instead of 4
8 wife's brother's daughters instead of 4

The woman now has:

4 brother's wives instead of 2
8 brother's sons instead of 4
8 brother's daughters instead of 4

Table 6 contains the relationships which are present or absent and

the quantity of individuals in each relationship for 1′, 2′, 3′, 4′ and 1″. Both the man and the woman relate to the identical individual. Thus there are always at least two relationships present in regard to each individual, one for the man and one for the woman.

It is to be noted that all of the relationships are present in 1′ and that all of the relationships for the nuclear family (line A) are present in all of the cases. However, the relationships for B are lacking in 2′, for D in 3′, and for both B and D in 4′.

When we look at F (grand total) for relationships, we find that 1′ and 1″ have the same quantity 6/32 but are different in C and E due to the double relationships in 1″. 2′ and 3′ have the same quantity of relationships 6/24 but are different in B and D. Thus none of the five contain the same constellation of relationships. When we carry this through all 32 forms of sexual mating and the resulting kinship systems we can then see that each of the 32 is a different kinship system.

When we compare the 5 cases in regard to the quantity of individuals we find that 1′ contains a greater quantity of individuals than relationships because there are 2 or 4 individuals in certain of the relationships. 2′ contains the largest quantity of individuals despite the fact that it contains fewer relationships than 1′. The constellation of relatives varies from one type of sexual mating to another, certain relationships are sometimes absent while others are consolidated.

From table 6 it appears that the greater the quantity of the force Sex (A) the fewer the relationships but the greater the quantity of individuals in some of the relationships.

As the quantity of A is increased there is a proportional decrease in the number of relationships. It appears that it is important to differentiate between the relationship and the quantity of individuals in the relationship.

SUMMARY

From the reported forms of sexual mating 6 forms have been presented as being basic: the Primary which is composed of one man and one woman of different families and 5 Secondaries: Sororate, Levirate, Wife's brother's daughter, Husband's sister's son, and Exchange. Combinations of two or more Secondaries result in a total of 26 possible Compounds making a total of 32 forms in all. These can be divided into a Prime series lacking Exchange matings and a Double Prime series involving Exchange. Within each series the forms may be represented

TABLE 6

Comparisons of Quantities of Relationships and Individuals

		I	II	III	IV				
		Q of Relationships	Man to his	Woman to her	Q Ind. per Relationship				
					1'	*2'*	*3'*	*4'*	*1''*
Nuclear Family	A	6	w		1	2	1	2	1
				h	1	1	2	2	1
			s	s	2	4	2	4	2
			d	d	2	4	2	4	2
			Total individuals		6	11	7	12	6
Affinal and Consanguineal Families	B	8	w si	si	1	0	1	0	1
			w si h	si h	1	0	2	0	1
			w si s	si s	2	0	2	0	2
			w si d	si d	2	0	2	0	2
			Total individuals		6	0	7	0	6
	C	8	w b	b	2	2	2	2	1–1[a]
			w b w	b w	2	4	1	2	1–1[b]
			w b s	b s	4	8	2	4	2–2[c]
			w b d	b d	4	8	2	4	2–2[d]
			Total individuals		12	22	7	12	6–6
	D	8	b	h b	1	1	0	0	1
			b w	h b w	1	2	0	0	1
			b s	h b s	2	4	0	0	2
			b d	h b d	2	4	0	0	2
			Total individuals		6	11	0	0	6
	E	8	si	h si	2	2	2	2	1–1[b]
			si h	h si h	2	1	4	2	1–1[a]
			si s	h si s	4	4	4	4	2–2[c]
			si d	h si d	4	4	4	4	2–2[d]
			Total individuals		12	11	14	12	6–0*
Grand Totals	F		Individuals		6/36	11/44	7/28	12/24	6/24–6
		6/32	Relationships		6/32	6/24	6/24	6/16	6/32

* Double Relationships.

		To the Man		To the Woman
1''	a	1—w b and si h si	—is the same individual	—b and h si h
	b	1—w b w and si	— " " " "	—b w and h si
	c	2—w b s and si s	— " " " "	—b s and h si s
	d	2—w b d and si d	— " " " "	—b d and h si d

in a diamond such that on one axis a progression from matings with a single female of one generation to multiple females of one generation to a single female from each of multiple generations to multiple females of multiple generations would be shown. On the other axis the corresponding progression for males would be shown and the whole 16-fold diamond could be superimposed on the corresponding one for the Double Prime series. Hypothetically one might expect changes in customary mating practices to follow the progressions indicated by such a model.

The 32 varieties of sexual matings were further analysed and seen to consist of invariant factors with quantitative variation, male(s) and female(s) of generation(s) of one family mated to those of another family. These can be expressed as a universal formula in which they are equated with a quantity (A). In general any change in A would be accompanied by a change in the form of sexual mating. Furthermore, a change in A would correlate positively with changes in the number of individuals in the sexual mating group, nuclear families, individuals in the nuclear family, individuals in the affinal and consanguineal nuclear families, individuals in the total population, relationships, individuals in the relationships, and families in the total population.

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BOOK REVIEWS

Genetica Medica. I^{um} Symposium Internationale, 6-7 Septembris MCMLIII. Edited by LUIGI GEDDA. Pages xxiii + 467, Istituto Gregorio Mendel, Rome, 1954. Price not indicated.

This book is a double first. It is the first volume of a new series, *Analecta Genetica*. And it is the proceedings of the First International Symposium of Medical Genetics.

The work opens with the remarks of Pope Pius XII, translations of which are given in Italian, English and German. Perhaps the sentence of the Papal remarks most significant to the geneticist *as* scientist is: "L'appréciation des questions purement spécifiques est laissée à la compétence de votre science" (p. xviii).

The proceedings of the symposium are divided into 12 *Relazioni* and 18 *Comunicazioni*. Two of the papers are in French (F), 5 in English (E), 7 in German (G) and 16 in Italian (I). Surnames of the authors, shortened titles, and an abbreviation of the language of the *Relazioni* are, in the order printed: Gedda, Medical genetics, I; Gianferrari, Longevity, I; Kallmann, Health and mental disorder, E; Franceschetti & Klein, Identification of heterozygotes, F; Lehmann, Endocrine diseases, G; Torrioli & Romei, Pernicious anemia, I; Waardenburg, Papilla leporina, vitiligo, and Leber's optic atrophy, E; von Verschuer, Longevity, G; Lamy & Bienenfeld, Discondrosteosy, F; Hanhart, Geography of human genes, G; Grebe, Pathology of the skeletal system, G; and Mollison, Hemolytic disease of the newborn, E.

The *Comunicazioni* are: Andreani, Conti & Monicelli, Germinal aplasia and hypogonadism, I; Arrigoni, Cresseri & Lovati, Polycystic disease of the kidney, I; Canziani & Riccobono, Tests for musical talent, I; Ceppellini, Aminoaciduria, I; Casa, Diabetes, I; De Biase, Dental anomaly discordant in MZ twins, I; Dell'Acqua, Somatic, biochemical, and physiological observations on MZ twins, I; Gatto, Drepanocytosis in Italy, I; Hoffer, Maxillo-facial anomaly, I; Jucci, Baldness, I; Korkhaus, Teeth and facial skeleton, G; Lombardi, Cardiovascular disease, I; Maltarello, Cooley's anemia in MZ twins, I; Oostenrijk, Poliomyelitis acuta anterior, E; Pavoni, Morganti & Avanza, Blood groups of Pesaro, I; Moench & Schaeuble, Laurence-Moon-Bardet-Biedl syndrome in twins, G; Stecher, Herberden's nodes, gout, and ankylosing spondylitis, E; and Testa, Histological study of twins, G.

The papers of this symposium vary greatly in purpose and scope. All are of interest and substance to medical genetics. My vote for the one paper of this group which might be of most general interest to the human biologist would go to E. Hanhart, Beiträge zu einer humangenetischen Geographie (pp. 165-187). Aside from the genes of the red blood cell antigens, previously little had been written in summary form on the population distribution of human genes.

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Clinical Versus Statistical Prediction: a Theoretical Analysis and a Review of the Evidence. By P. E. MEEHL. Pages vii + 149, University of Minnesota Press, Minneapolis, 1954. $3.00.

In this slim, attractively produced book Dr. Meehl has set out to examine a burning problem in clinical psychology. The first part is devoted to a statement of the problem, to the examination of logical issues, to the reactions of other writers, *e. g.*, Sarbin, and to an analysis of clinical intuition. The later chapters are concerned with empirical evidence on the relative effectiveness of the two methods and with problems of quantification of clinical observations.

At the commencement Dr. Meehl points out an important distinction between the *discriminative* and the *structural* use of statistics. The former is represented by tests of significance. The latter is concerned with more complex psychological constructs, used to 'explain' behavior. As Dr. Meehl correctly emphasizes, "the prototype of this use of statistics is factor analysis." The term "actuarial," which Dr. Meehl uses in his chapter headings as opposed to "clinical," is intended to include this more potent use of statistics and not merely the blind, non-psychological, non-theoretical use of actuarial statistics.

The definition of "clinical," however, is not reached so early or clearly. The reader finds himself asking: "Do we mean conclusions reached without conscious awareness of the logical or mathematical processes?" "Or without awareness of all the observations being taken into the judgment?" "Or by use of facts and principles precisely similar to that of the psychometrician but involving *mental* calculation instead of formal computation?" "Or by use of mathematical relationships not yet employed by statistical psychologists?" At times Dr. Meehl seems to be toying with this last meaning, which would be a quite unwarranted use, for *any* conceivable functional relationships of observations and predicted criteria can be equally employed, implicitly by the clinician and explicitly by the personality statistician. It would be ridiculous to restrict the latter, for example, to first order equations or to non-pattern considerations. Dr. Meehl's proposition that the statistician may also use "prediction from a class" indicates that he rejects this restriction. But reference, for example, to the Pattern Similarity Coefficient, r_p, applied to patterns of functionally unitary factors, would have illustrated a realm apparently little known to clinicians (as the writer later states), and would have left no remaining doubt of the writer's theoretical position.

At times Dr. Meehl flirts with a still more untenable definition of the clinical method, when he proceeds in his argument to represent the clinical method by a mature, full-fledged psychotherapist and the actuarial method by a young woman clerical worker substituting figures in given equations! (The impression is somehow given that she barely graduated from high school and is rather weakminded.) If we are to compare the two methods, let us at least have trained psychologists in both positions, each fully aware of whatever psychologically predictive principles can be applied to the observations! A similar sop to the clinician is gratuitously given by Dr. Meehl when he quotes Dr. Reik's account of a remarkable and (as events proved) true inference drawn by Dr. Reik from a very small symptom, and asks what the statistician would do in such a case. I'm afraid the statistician would first ask Dr. Reik to try to remember all the unrecorded instances where an inspired guess proved *wrong*! To the reviewer the only satisfactory definition of the clinical method is that it proceeds in part upon unconsciously perceived data and in part by inexplicit or unconscious reasoning, *i. e.*, "intuitive" application of logical and mathematical relations. In so doing, it uses wholistic and pattern formulations which, prior to the development of factor analysis and pattern calculation, were not used by the psychometrist, and are admittedly *still* not used by most psychometrists "trained" for clinical work.

On reaching the empirical treatment Dr. Meehl musters no fewer than nineteen studies—all very interesting reading—in the great majority of which he ultimately concludes that the statistical predictions have been significantly better than the clinical ones. Few of these studies have been even tolerably well designed to answer all that could be answered.

Little attempt has been made to see that the clinical and the statistical representatives have reached equal maturity of training in their studies, or even that they are supplied with the same data or testing time of the subjects, or that the area of possible prediction is well sampled; but Dr. Meehl shrewdly appraises these uncontrolled variables. Actually, as he points out, in these unequal competitions it is always the clinician who has been supplied with more time and data and who yet fails to reach the same efficiency of prediction. Many insufficiently known findings come up in this evaluative study, *e.g.*, Baron's finding that in the Rorschach experiment "neither a mechanical use of the usually mentioned signs, nor a subjective sorting by four Rorschach experts (with access to the protocols) showed any correlation with therapeutic outcome."

The final chapters on quantification with use of patterns, etc., are cut very short, just when the statistician would be interested in resolving alternative possible conceptualizations of the significant predictions from patterns made by Dr. Meehl and others. In any case, due to the instability of mere individual "items" in tests the psychologist is going to be more interested in patterns based on psychologically meaningful temperamental and dynamic *factors* than in patterns in arbitrary *items*. With so much work still needing to be done on primary personality factors and the trying out of simple linear predictions from these psychological entities, it is intellectually very easy but strategically premature to be insisting that additive relations are not enough. As Meehl well says, "There is a lot of loose talk around these days about non-linear relations. I do not doubt that there are a large number of such in the behavior domain, but we ought not to browbeat the statisticians with this phrase until we know more about where these non-linear dependencies occur and how much they pay off predictively over and above the much-maligned linear regression system."

A careful theoretical and empirical evaluation of this problem has been long overdue. It is hard to imagine anyone better qualified by combination of clinical and statistical viewpoints than Dr. Meehl to tackle this involved question. Certainly clinicians are likely to give more serious attention to these unfavorable findings when they come from a person of established reputation in their own realm than from a clinically uninterested statistician. Actually, in the reviewer's opinion, Dr. Meehl has maintained the balance very fairly, but perhaps with a little more charity to his kin than others! The sooner this painful self-knowledge is accepted by the hard pressed clinician, the sooner will true remedies, in terms of better research-based tests and more insightful prognostic procedures, become possible.

RAYMOND B. CATTELL

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Human Heredity. By JAMES V. NEEL AND WILLIAM J. SCHULL. Pages 361, University of Chicago Press, Chicago, 1954. $6.00.

This is a general textbook of human heredity with emphasis on methodology. We think it is outstanding and certain to play a large role in the training of future human geneticists. It is intended for readers who wish to learn not only accumulated facts and theories, but research procedures as well. Much of the book is devoted to various ingenious devices of Weinberg, Fisher, and others for handling difficult problems in Mendelian ratios, linkage, etc. that would be simple in animals where experimental matings are possible. The principal tool is the maximum likelihood method, which is introduced early and used consistently throughout the book. We think this is a good idea, for although it is debatable whether this is always the best method, there is the real merit of giving the student one asymptotically efficient procedure that is applicable to a wide variety of problems.

The authors start out on an optimistic note by pointing out some of the advantages of man as an object of genetic study, rather than following the usual practice of deploring his obvious disadvantages. Then comes a very short chapter on cytology. The nature-nurture problem is introduced early and discussed throughout the book with no attempt to hide its complexity. There is a clear and balanced presentation of the methods and results of mutation rate study without undue emphasis on the authors' own minority view that average rates are high. After several chapters on formal genetics the book ends with sections on epidemiology, counseling, forensic genetics, and eugenics.

The chapter on physiological genetics is good and we should like to see it expanded in future editions. The section on blood groups is surprisingly brief, but perhaps this isn't too serious because of the excellent books available on this subject (*e. g.*, Race and Sanger). The section on testing Mendelian ratios with various types of ascertainment bias is the clearest account we have seen. But we think there is a serious omission; no method is provided for the case where ascertainment is incomplete but known, and since the simply applied Weinberg proband method is appropriate and of fairly high efficiency we should like to have had it included. The chapter on linkage gives only the Penrose sib pair method. Here is one of the few places where we believe the authors could have been more critical by pointing out the liability to error and the low efficiency of this method. Unfortunately, methods of detecting and measuring linkage are complex, but perhaps the gift the authors have shown for clear writing could be used to present some better methods. The authors regard as the main purpose of linkage study its possible use in prognosis. We should prefer to emphasize linkage relations as a research tool for distinguishing between phenotypically similar, but genetically different entities or for calling attention to such heterogeneity. The chapter on twin studies is particularly good in pointing out pitfalls in interpretation for the uncritical. Our only complaint is that, in using Holzinger's H as a measure of heritability, they fail to note that this is quite different from heritability as used by animal and plant breeders. Finally we like the chapters on counseling and eugenics, perhaps because we share the authors' reservations.

Although the book is very modern in its approach, it is already out of date in some areas. This is, of course, a statement about the rate of advance in knowledge rather than a criticism of the authors, who, by being responsible for some of the advances, have shortened the useful life of their book. Some new works that are not included are: the relation of thalassemia and sicklemia to malaria resistance, evidence for mutation at the MN locus, new work on hemoglobins, possible position effects and the f antigen in the Rh series, new evidence for selection in the ABO blood groups, and new factors governing prothrombin formation. We are already looking forward to a second edition.

In subject-matter this volume is similar to Stern's *Principles of Human Genetics*. It differs in being more concise, in having a greater emphasis on research methods, and in assuming more knowledge of genetics, statistics, and differential calculus on the part of the reader. The authors make no apology for this. They say: "The complexities of the study of human heredity are such that knowledge of certain branches of mathematics is no less essential to the serious student of human heredity than to the astronomer or physical chemist. The text which attempts to disguise this fact is, in the long run, doing the student a disservice." Here, again, we agree emphatically.

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Atlas of Men. A Guide for Somatotyping the Adult Male at All Ages. By W. H. SHELDON, with the collaboration of C. WESLEY DUPERTUIS AND EUGENE MCDERMOTT. Pages xvi + 357, Harper, New York, 1954. $10.00.

When the mariners of the days of Vasco da Gama and Columbus left their native shores and trusted to the wind of discovery they brought back with them a variety of hard-earned treasures. There was gold perhaps, and strange animals, and sometimes overcredulous tales; but for those who came after them the most important thing they brought was an atlas, a map of countries other than their own. And so perhaps with all exploration; each of us has to sail on his own private voyage from his own private island to meet others inhabiting other islands which may seem strange or engulfing or hostile. For this more personal journey, we need an atlas too, because if we know something of the natures of people different from ourselves and expect of them behaviour also different we shall be much less likely ourselves to be frightened or hostile, righteous or overbearing. Just as the scientific study of primitive peoples has had the effect of increasing our understanding and sympathy with them and has enabled us to learn more about ourselves, so the scientific study of the differences in constitutional make-up of people in a single culture can lead to increased understanding and sympathy between people of different temperament, physique and aptitude. In this, Sheldon's *Atlas of Men* has perhaps the usefulness, partial and imperfect, that the first primitive maps of the ocean had for the early seafarers.

The book is primarly an atlas of photographs, and as such it must be judged. It consists of 1175 sets of somatotype photographs, beginning with the 117 and progressing through the ones in endomorphy by the steps 126, 127, 136, 145, 154, 162, 163, 171 and 172. Then come the two's in endomorphy arranged in a similar order; then the three's, and so on. The material on which the atlas is based is quite staggering. During the last 20 years Sheldon and his collaborators have photographed and somatotyped some 46,000 men, of all ages (from 18 to 65). On the first 4000 the procedure of somatotyping and the scales of the three components were standarized: this formed the basis for the *Varieties of Human Physique*, published in 1940. Now, in the more extensive material, 88 somatotypes have been found, or 505 somatotypes if 13-point rating scales are used for each component instead of the original 7-point scales. Each of these 505 physiques is presented in the atlas, and nearly all of them at two or three different ages—typically about 20, 40 and 60 years. The somatotype of each of the five regions of the body is given for each picture, so that the dysplasia of each is also recorded. In addition a list is given of pictures which together form scales for androgyny of physique and for texture-component.

The pictures themselves measure about 3½″ x 4″, that is, about two-thirds of the size at which they were originally taken. Some loss of detail is brought about by this reduction, but much more by the halftone process itself: one of the minor frustrations of all workers in this field is to see their most cherished photographs, which in the original showed every faint muscle shadow and every individual hair, reduced on publication to a uniform shapeless grey (with X-rays the situation is still worse). It is idle to pretend that these reproductions replace for the actual somatotyper a key file of real photographs, though the publishers have probably done as well with them as is possible at present. They are certainly a considerable improvement over those in the *Varieties of Human Physique*. Inevitably in such a huge collection the quality of the originals varies considerably, both as to photographic and posing technique. On the first page, for example, No. 2 shows a lot of detail that in No. 4 has been quite obliterated, and the pose of No. 762, compared with that of No. 772, leaves a good deal to be desired.

These, however, are the technical criticisms of the fellowcraftsman. Let it be said at once that this atlas is a magnificent achievement, a feat of sustained and meticulous endeavour which has no parallel whatever in the previous history of the subject. Even to the casual glance the range of variation in human morphology displayed cannot fail to be awe-inspiring. Turn, for example, to page 71 and compare the 172 and the 216. Or contemplate Nos. 369 and 1078 (a 271 and a 6½ 2½ 1½), who are almost identical in height and weight but totally dissimilar in shape and in tissue composition. Look at No. 1038, and then No. 138. "If sometimes you find yourself doubtful about the ultimate fate of man" writes Sheldon "and in need of reassurance as to the vastness of his adaptability, try to contemplate the enormous range of adaptive potentiality that must inhere in such radically different somatotypes as the 651 and the 145. Then recall that these two represent only two points in a far-flung, continuous extension in three dimensions. Perhaps we do well to carry the great range of somatotype variation that this present culture appears to afford. Therein may lie the best insurance against an untimely ending of the human play."

With the pictures goes a page of text for each somatotype. This includes a chart of the particular somatotype's position on the conventional somatotype distribution diagram, a graph of the average height/$\sqrt[3]{\text{weight}}$ at each age from 18 to 65, and a table of weight-for-height at each age. These graphs and tables are invaluable, and with them we have for the first time standards of weight for height which are not nonsensical. They may be too detailed for present clinical use, but certainly from them broader categorisations with appropriate standards could quite easily be made. They are admittedly based, as is all the atlas and the somatotyping, on cross-sectional material, and Sheldon is rightly insistent that this makes them subject to revision when longitudinal data to replace them become available. Clearly we need pictures of the *same* 354 at 20 and 40 and 60 and not pictures of persons aged 40 and 60 who, in the somatotypers opinion, probably at 20 were the same somatotype as the 354 20-year old. Those involved in the field should now already be planning another atlas on longitudinal lines, which will need yet more determination, and yet more sympathetic farsightedness from those in control of funds and facilities. They will also be planning to take X-rays as well as photographs, skinfold thicknesses as well as weight and height, and a whole variety of physiological and psychological measurements. But that is another step forward, and here we are concerned with the nature and imprint of the first step only.

Below the tables of weight and height for each somatatype is half a page of general comment. It seems to me a pity that this does not include the detailed physical description of the somatatype given in the *Varieties of Human Physique,* which instead has to be used in conjunction with the atlas if the best use is to be made of the pictures. The general comments however, as one would anticipate in any book of Sheldon's are always interesting and lively, though he specifically warns the reader that they are to be taken as " impressions provocative of new research . . . and not confused with established fact." They reveal in particular an interest in longevity and in the applications of somatotyping to clinical medicine and psychiatry.

One minor feature of the book will doubtless come in for a disproportionate amount of comment. Sheldon has given an animal totem to each somatotype, an amusing echo of Aristotle and Della Porta. For each somatotype there are a few lines in italics ostensibly describing the animal, but endeavouring to refer obliquely to some aspect of the somatotype about to be described. Sheldon calls these italicised lines "self-evidently an accompaniment in a minor key to be read or omitted accordingly to the reader's taste." The metaphor is apt; not all listeners are able to distinguish the major from the minor mode, and those who cannot had much better leave the italics alone. They should perhaps bear in mind, however, that a disinclination to smile does not of itself signify the taking of a subject *au sérieux*, and vice versa. Inevitably, some of the totems are far-fetched and largely beside the mark, but in others the short and telling phrase—"killed by a careless gesture, but for all that they can sting resolutely"—reveals the sharp pen of the born novelist.

Preceding the atlas proper there are some thirty pages of introduction. In them are discussed, with equanimity and a wit quite innocent of the rancourous overtones which marred some pages of an earlier book, the history of somatotyping, the effect of age and nutrition on somatotype, the scales for use in somatotyping women, and the relative frequency of each somatotype. One remark here, however, is quite misleading. Sheldon implies (pp. 5, 14) that the three somatotype components are orthogonal. This is by no means the case; they are negatively interrelated to the extent that (judging from the figures of the earlier book) only between one-half and one-third of the variance of each component is left when the other two components are held constant. It is a pity that joint frequency distributions of the components are not given in the atlas; the frequency distributions for single components are there and show that mesomorphy and ectomorphy are very nearly normally distributed, while endomorphy has a long tail of high values and a shortage of low ones (that is, positive skew).

This obliquity of the components is one of the arguments against the usefulness of somatotypes as a method of classification. It is not a very strong argument, for it may really be that the somatotype components are more than statistical abstractions and have roots in physiological and genetical mechanisms. Though biometrically it is somewhat harder to handle oblique than orthogonal factors, the ultimate choice between them depends on the biological and not the statistical background. Only further experiment and more knowledge can decide the ultimate fate of Sheldon's classification; data alone will do so, not debate.

Meanwhile "by taking a careful weight history and using the height-weight criteria included in this volume, by matching your standardised photographs against these plates, and by studying the published checklists, it will be found possible to somatotype." Of one thing Sheldon may remain certain: this atlas is a landmark in the study of human physique and behaviour that will not be submerged by the tides of many decades. Somatotyping has changed the study of physique immeasurably and brought an order and precision that the field never knew before, but which now it will keep whatever change the future may bring. Somatotyping has also been a major influence in bringing about the present renewed interest in human constitution and human biology amongst medical men and biologists. These are great achievements for Sheldon and Dupertuis and their later colleagues to have accomplished, and they have put us all most surely in their debt.

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Perspectives in Physiology: An international symposium. Edited by ILZA VEITH. Pages 171, American Physiological Society, Washington, D. C., 1954. $3.00.

At the XIXth International Physiological Congress, held in Montreal in 1953, sixteen leading representatives of physiological science were brought together to exchange thoughts on the future and limitations of physiology and to present a picture of the past and present state of physiology in their respective countries. The essays in this little book are the presentations made in the introductory sessions of the symposium.

The book is divided into two sections, the first on "Limitations and Future," the second on "The Present Scene." The tone of the former is philosophical, of the latter documentary. It is a varied fare. Some of the essayists are preoccupied with the problems of communication and organization posed by the rapid expansion and fragmentation of physiological science. A few broach the more difficult question of *quo animo.* Thus Homer Smith of the United States exhorts us to again become *physiologoi* in the original Greek sense, concerned with a knowledge of nature in any of her aspects. And Alexander von Muralt of Switzerland says, in regard to the question: what is life?, that although we have dispensed with the old vitalism we are still faced with an ultimate restriction and must exclaim with Du Bois-Reymond "ignoramus, ignorabimus." For K. M. Bykov of Russia the lines along which physiological science should be developed are those defined by I. P. Pavlov. To Bykov it was Pavlov who introduced the "synthetic, materialistic" approach in contrast to the "analytic, idealistic" approach of Western Europe.

In the section describing the present status of physiology in various countries the essays range from catalogs of personnel and laboratories to dispassionate analyses of the toll taken by war and political upheaval.

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The Biologic Effects of Tobacco. Edited by ERNEST L. WYNDER. Pages xiii + 215, Little, Brown and Company, Boston, 1955. $4.50.

Smoking is a typically complex problem of human biology. Of its many aspects the present volume covers the chemical and pharmacological fundamentals, and the medical implications (allergies, cardiovascular system, gastrointestinal tract, and neoplastic diseases). The chapter devoted to the "cause and effect" in epidemiological research should help to clarify the important concept of causation in the etiology of disease. E. C. Hammond defines a cause of a disease as a totality of conditions that is sufficient to produce it. Because of the ever-present individual variability in reactions to a given set of environmental conditions, a statistical interpretation of causation is favored and a causative factor is defined as "any condition which, in a given situation, increases the probability that a specific event will occur."

The compilation of the volume was stimulated by the current debate on the theme "To smoke or not to smoke." The 8 authors present a critical review of the available evidence, together with detailed references to original investigations. The work will serve well to bring the medical and scientific professions up to date on the subject.

While on some points our present knowledge is fairly definitive, on other points additional research is called for. Acute effects in normal individuals, long-term role in the etiology of degenerative diseases, and effects in patients with specific diseases must be clearly differentiated. The incidence of diseases suggesting association with smoking varies tremendously. Except for a few, very rare diseases, the specific reactions (including reduced peripheral blood flow) are not involved in the etiology of abnormalities of the cardiovascular system, even though the use of tobacco influences unfavorably the clinical course of established diseases, *e. g.*, of hypertension. On the other hand, lung cancer, in the etiology of which tobacco smoking (especially cigarette smoking) is held to play a definite role, is among the most common causes of cancer deaths in males (p. 110).

To the human biologist, tobacco consumption is of interest as one of the facets of the mode of life that exhibits large differences between individuals, between the sexes, and between geographic areas. To some, smoking is a highly treasured means of relaxation, while others regard it as an annoyance or a "sin." In any comprehensive account, the positive value attached to tobacco consumption can not be disregarded. There are on record verified observations that prisoners on the verge of starvation traded their meager nourishment for a few puffs. Some patients, suffering from diseases of the peripheral blood vessels, warned repeatedly against using tobacco, will continue to smoke in spite of the fact that smoking brings on agonizing pain from gangrenous ulcers and necessitates multiple amputations.

A cigarette may serve as a protest against parental control, a cigar as an outward symbol of social status. Smoking, as a facet of the overall mode of life, has rich and inadequately explored implications for cultural anthropology, the study of personality development, and, in its abnormal aspects, for psychiatry. How well substantiated is the impression that heavy smokers are tense, "nervous" individuals? How effective a relief of "nervousness" does smoking actually provide? Is nicotine the only factor in the enjoyment of smoking? As an "oral phenomenon," how is smoking related to appetite and weight control? Observations are available indicating that cessation of smoking among excessive smokers, who were underweight and complained of anorexia, leads to a gain of weight. However, there are no adequate data for samples of "normal" individuals.

More generally, we may ask: In what biologically relevant ways do different categories of smokers differ from non-smokers? To this question the present volume gives answers limited largely to considerations of disease. Scientific clarification of such problems as the relationship of tobacco consumption to personality, body weight and body composition must await further research. However, further work is needed on such basic problems as the carcenogenicity of tobacco smoke, the carcenogen being so far unidentified, and on the very nature of the association between smoking and lung cancer (*cf.* J. Berkson, Proc. Staff Mayo Clinic, *30:* 319-348, 1955).

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BOOK NOTICES

FUNDAMENTALS OF RADIOBIOLOGY. By Z. M. Bacq and P. Alexander. xii + 389 pp. Academic Press, Inc., New York, 1955. $6.50. In the age of nuclear fission, the use of radiation in the treatment of cancer and the problem of avoidance of serious exposure hazards to health and life itself provide a strong practical stimulus to research in this field, requiring the pooling of the resources and methods of the physicist, chemist, biologist and clinician. The volume aims at an overall survey of the present, widely scattered information. Beginning with the physics of ionizing radiations, the survey ends with observations on human beings. Genetical phenomena, biochemistry of irradiated living organisms, and the action of the endocrines (pituitary, adrenal glands) in radiation sickness are also considered. Close to 1000 papers are quoted.

HISTORY OF THE PRIMATES. By W. E. Le Gros Clark. 119 pp. British Museum, London, 1954. 4 Shillings. This is a fourth edition of a readable and authoritative introduction to the study of the fossil man.

THE LUNG. By Julius H. Comroe, Robert E. Forster, Arthur B. Dubois, William A. Briscoe and Elizabeth Carlsen. viii + 219 pp. The Year Book Publishers, Inc., Chicago, 1955. $5.50. A book on clinical physiology and pulmonary function tests, written for physicians and medical students. Its aim is to explain in simple words and diagrams those aspects of pulmonary physiology that are important to clinical medicine.

THE HUMAN ORGANISM. By Russell Myles De Coursey. vii + 550 pp. McGraw-Hill Book Company, Inc., New York, 1955. $5.75. An elementary introduction to human anatomy and physiology, richly illustrated, with a glossary, suggested readings (principally books), and questions appended to each chapter.

RECEPTORS AND SENSORY PERCEPTION. By Ragnar Granit. xi + 369 pp. Yale University Press, New Haven, Connecticut, 1955. $5.00. These Silliman lectures, presented by one of the most prominent workers in the field, are a reflective "comment on the achievements of a quarter of a century of work by electronic methods on the special senses." The author had a significant share in the experimental investigations concerned with senses as interpreters of the external world. At the same time, the action-current analysis of the function of sensory organs was treated as a part of general neurophysiology. Of particular interest is the last chapter on sensory discrimination and integration. With its 50-odd pages of references, this volume is the best available summary of a fascinating but highly specialized field.

LABORATORY INSTRUMENTATION IN PSYCHOLOGY. By William W. Grings. vi + 282 pp. The National Press, Palo Alto, California, 1954. $4.75. Intended as introduction to the subject, at a moderately advanced level. Has three general chapters (recording systems, timing and counting, biolectrical phenomena), three chapters on special senses (audition, vision, other senses), and chapters on human learning and perception. Gives references to more specialized and advanced sources. A more comprehensive treatment of the subject-matter in the future editions would be useful.

PSYCHOMETRIC METHODS. By J. P. Guilford. ix + 597 pp. McGraw-Hill Book Co., Inc., New York, 1954. $8.50. The pioneering first edition (1936), addressed to graduate students of psychology, combined the specific problems of psychological measurement (psychophysics, psychological scaling methods, mental tests) and the more general statistical treatment of quantitative data. With the statistical methods presented in *Fundamental Statistics in Psychology and Education* (1950), the second edition of *Psychometric Methods* is focussed on measurement in psychology, its theory, procedures, and the more specific statistical operations (such as the derivation of a factor matrix from a correlation matrix by factor-analysis methods).

VITAMINS IN THEORY AND PRACTICE. By Leslie J. Harris. xxii + 366 pp. Cambridge University Press, London, 1955. $6.50. This book, first published in 1935, grew out of popular lectures given at the Royal Institute, London, in 1934. The aim of the fourth edition remains to present, in a readable form, an up-to-date story of vitamin research.

GENETICS AND HUMAN HEREDITY. By J. Ben Hill and Helen D. Hill. xiv + 526 pp. McGraw-Hill Book Co., Inc., New York, 1955. $6.50. A text designed for students in elementary genetics. Examples and illustrations are drawn about equally from the plant and animal kingdoms.

HUMAN PHYSIOLOGY. By Bernardo A. Houssay, Juan T. Lewis, Oscar Orías, Eduardo Braun-Menéndez, Enrique Hug, Virgilio G. Foglia, and Luis F. Leloir. xvi + 1177 pp. McGraw-Hill Book Co., Inc., New York, 1955. $12.00. The appearance of the second edition, within 4 years, of this comprehensive textbook indicates that it has found a firm place in a highly competitive market. Written for medical students, it will serve well as a reference book for physicians and for scientists interested in facts and fundamental principles of modern physiology. The authors are teachers as well as productive investigators. The fact that they did not have to depend for describing the major portion of the *globus physiologicus* solely on literature (English, as well as French, Italian, German and Spanish) but could draw on first-hand experience, constitutes one reason for the appeal of this book.

STATISTICS AND MATHEMATICS IN BIOLOGY. Edited by Oscar Kempthorne, Theodore A. Bancroft, John W. Gowen, and Jay L. Lush. ix + 632 pp. The Iowa State College Press, Ames, Iowa, 1954. $6.75. The present volume grew out of the need for synthesizing concepts and methods of biology with those of mathematics (development of quantitative theories) and statistics (economy of description, including condensation of observations, prediction). The 44 papers were presented at a biostatistics conference, held at Iowa State College in 1952. They fall into 5 groups: general biometric principles and procedures, changes in population numbers, estimation of population size, evaluation of biological responses, and genetical analysis of populations.

THE JOSIAH MACY, JR. FOUNDATION 1930-1955: A REVIEW OF ACTIVITIES. xi + 174 pp. Josiah Macy, Jr. Foundation, New York, 1955. A review of activities designed to "initiate, stimulate, develop and support scientific investigations of the fundamental aspects of health, of sickness, and of methods for the relief of suffering." The Foundation has played a significant role in the advancement of knowledge in medical and social sciences. Most important, perhaps, it provided liberal support for interdisciplinary collaboration essential for the integration of knowledge obtained in the separate sectors of the study of man.

ELECTROCHEMISTRY IN BIOLOGY AND MEDICINE. Edited by Theodore Shedlovsky. xii + 369 pp. John Wiley and Sons, Inc., New York. Chapman and Hall, Ltd., London, 1955. $10.50. Living organisms are able to transform chemical eneregy into electrical signals. The study of these signals provides important fundamental information about the normal function of nerves and muscles, and aids the physician in the diagnosis of the disordered function of heart and brain. The volume grew out of the 1953 symposium sponsored by the Electrochemical Society. The 18 papers were contributed by physicists, chemists, biologists, and clinical investigators. They present, in a highly technical fashion, the results of electrochemical researches on membranes, cells, ionic activity, polarography of plasma proteins, electrocardiography and electroencephalography.

BIOMATHEMATICS: THE PRINCIPLES OF MATHEMATICS FOR STUDENTS OF BIOLOGICAL SCIENCE. By Cedric A. B. Smith, xv + 712 pp. Hafner Publishing Co., New York, 1954. $13.50. This is the third edition of W. M. Feldman's *vademecum* on the application of mathematics to biology. Numbers, shapes and chances are all considered in the quantitative description of biological phenomena, even though here the emphasis is on arithmetic and geometry rather than on statistical methods. From a brush-up on elementary rules and tricks of arithmetic, the reader is led all the way to the mathematics of the rates of change, various ways of solving equations and the study of matrices.

ESSENTIALS OF BIOLOGICAL AND MEDICAL PHYSICS. By Ralph W. Stacy, David T. Williams, Ralph E. Worden and Rex O. McMorris. xiv + 586 pp. McGraw-Hill Book Co., Inc., New York, 1955. $8.50. A well organized textbook in a rapidly growing field, intended for use in courses on biophysics offered to students of biology (as one of the foundation blocks of their science), physics (as a facet of applied physics), and medicine (as a basis for some forms of therapy). It contains the following sections: Fundamental concepts, Mechanical systems (bones, muscles; effects of high accelerations on human organism), Heat, Sound, Light, Gas physics, Flow of fluids, Electrical phenomena, Nuclear physics, and Theoretical biophysics. Six appendixes.

GENERAL ENDOCRINOLOGY. By C. Donnell Turner. xi + 553 pp. W. B. Saunders Co., Philadelphia, 1955. Endocrinology is presented as a basic biological science, with internal secretion regarded as a mechanism operating throughout the whole range of plant and animal life and physiologically important during the developmental period as well as in adult individuals. With reference to the separate glands of vertebrates, anatomical features, biochemistry of the particular hormone, and the physiological effects are presented in turn. Photographs illustrating typical endocrine disturbances in man and animals are provided, but the clinical aspects of endocrinology are not treated extensively. In comparison with the first edition (1948), the principal addition is a chapter on adaptive reactions to stress.

THE MAMMALIAN FETUS: PHYSIOLOGICAL ASPECTS OF DEVELOPMENT. Cold Spring Harbor Symposia on Quantitative Biology, Vol. 19, xii + 226 pp. Biological Laboratory, Cold Spring Harbor, New York. $8.00. The primary aim of the conference was to evaluate the present knowledge about the morphological, physiological and biochemical factors affecting the mammalian, especially the human, embryo. Emphasis was placed on mechanisms involved in the somatic and visceral development essential for normal living before and just after birth, even though pathology (such as radiation effects) was also considered. Twenty-seven papers by American and European authors, with synthetic chapters (biochemical and physiological evaluation) and a review of implications for the management of the newborn infant.

THE MICMAC INDIANS OF EASTERN CANADA. By Wilson D. Wallis and Ruth Sawtell Wallis. xv + 515 pp. University of Minnesota Press, Minneapolis, Minnesota, 1955. $7.50. The field work among the Micmac was carried out in 1911, 1912, 1950 and 1953. The purpose of the early work was to obtain a detailed ethnographic account. The recent studies were focussed on cultural changes.

PHYSIOLOGICAL ACOUSTICS. By Ernest Glen Wever and Merle Lawrence. xii + 454 pp. Princeton University Press, Princeton, New Jersey, 1954. $10.00. Hearing involves a series of events—mechanical, electrophysiological (electrochemical), and psychological (perception of sound). The volume is a highly competent presentation of the initial, mechanical segment of this series, focussed on the function of the middle ear as an acoustical transformer and on sound conduction in the cochlea. Clinical applications (conductive deafness, otosclerosis) are considered and the present status of auditory theory is reviewed. Eighteen pages of references.

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